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MALARIA

In this section abstracts are arranged as far as possible in the following order:—Human malaria—*epidemiology, aetiology, transmission, pathology, diagnosis, clinical findings, treatment, control;* Animal malaria—*monkeys, other animals, birds.*

DIAZ MARIN, J. El problema del paludismo en el continente africano. [**The Malaria Problem in Africa**] *Med. Trop.* Madrid. 1959, May–June, v. 33, Nos. 5/6, 297–308. [19 refs.]

A general review.

DAGGY, R. H. **Malaria in Oases of Eastern Saudi Arabia.** *Amer. J. Trop. Med. & Hyg.* 1959, Mar., v. 8, No. 2, Pt. 2, 223–91, 24 figs. [Numerous refs.]

The Qatif and Al-Hasa oases are large, well watered, rich agricultural tracts of 7,000 and 30,000 acres of cultivable land respectively, carrying 150,000 and 160,000 people and constituting major components of the Saudi Arabian economy. The people, housing, agriculture, irrigation practices and climate are described in detail, and malaria is shown to have been very severe, clearly of an unstable type causing severe morbidity, evidenced in both the seasonal and more widely spaced epidemics.

Very full records are given of a number of malaria surveys of these areas before the institution of control. The anopheline fauna included *Anopheles stephensi, A. fluviatilis, A. sergenti, A. pulcherrimus, A. multicolor* and *A. coustani*, while that of the country and its neighbours includes these and an additional 19 listed species. The characteristics of each of the local species are described with particular emphasis on those of *A. stephensi*, which is considered to be the sole vector. The seasonal distribution of this mosquito is particularly considered and a picture is built up which relates its number to the varying temperature and humidity throughout the year, making predictions of its prevalence possible. Malaria control was started in these oases in 1948 and was carried on, with intermissions which are recorded, until 1955. The DDT spray had originally apparently obtained a total control of the malaria, but it became evident towards the end of this period that the disease was recurring and that anophelines were persisting in material numbers on treated

surfaces. Enquiry then showed that the mosquito had become resistant and dieldrin spraying was substituted for the previous DDT applications. Full control of malaria was then re-established.

The author considers, however, the nature of the problem facing the authorities should resistance to this insecticide become selective, and reviews the possibilities of control by traditional methods of larvicides and the control of breeding places by water management, drainage, filling and such like measures. [It would however appear rational to aim at the eradication of malaria while this is possible with the insecticides now in use.] *G. Macdonald*

METSELAAR, D. **Two Malaria Surveys in the Central Mountains of Netherlands New Guinea.** *Amer. J. Trop. Med. & Hyg.* 1959, May, v. 8, No. 3, 364–7. [16 refs.]

It has long been maintained that malaria is not transmitted in New Guinea at altitudes over 1,200 m. (3,937 ft.) and that isolation of population groups by tribal warfare prevented the distribution of the parasites, and evidence to the contrary is at times disbelieved. The author surveyed some groups of people who had until very recently been in isolation in a stone age culture and lived in or above two valleys, the bases of which lie at 1,600 m. (5,250 ft.). Numerous specimens of *Anopheles farauti* and *A. koliensis* were collected. In one of these valleys there had been a recent epidemic of malaria in which at least 16 people out of about 1,000 had died; the people living in its base showed a parasite rate of 38%, those somewhat up the slope from the base had a rate of 15%, while in two communities on the ridge above, the rates were 48% and 7%. These variations are attributable to the site of the home, near or remote from anopheline breeding places, and also to the habits of the people, those most distant from the valley having to sleep in it on their regular journeys to it for salt, while nearer neighbours returned at night to their safer villages.

An interesting trader community was also studied. It is a traditional intermediary group which handles all merchandise, such as it is, and in this trade migrates continually from village to village. 5 malaria infections were found in 41 people examined, and such a group could clearly act as an intermediary for the carriage of malaria from village to village, just as for other articles.

The author considers that malaria has from time immemorial been carried at altitudes exceeding 1,200 m., and perhaps commonly up to 1,700 m. (5,578 ft.). The limiting factor is probably temperature, and not anopheline prevalence as has been suggested, and near this usual upper limit there are probably considerable areas of anophelism without malaria.

[See also this *Bulletin*, 1959, v. 56, 269.] *G. Macdonald*

ARCHIBALD, H. M. **Influence of Maternal Malaria on Newborn Infants.** *Brit. Med. J.* 1958, Dec. 20, 1512–14.

It has long been known that in women infected with *Plasmodium falciparum*, the placental sinuses on the maternal side are to be found packed with schizonts during the latter half of pregnancy. There has, however, been sharp controversy on the effect of this on the infant *in utero* and in neonatal life.

Whereas on the one hand BLACKLOCK and GORDON [this *Bulletin*, 1926, v. 23, 130] from Sierra Leone stated that there was "proof of a very complete association between maternal infection of the placenta and death of the child *in utero* or within a seven-day observation period after birth", on the other hand GARNHAM [*ibid.*, 1949, v. 46, 1113] from experience in Kenya expressed the opinion that "malarial infection in the mother seldom resulted in stillbirths or in the death of the infant in the first week". The author has re-examined this problem in Northern Nigeria in a large number of mothers delivered in institutions (who therefore represented a selected sample of the community) by examining for plasmodia capillary blood and placental smears taken at parturition and recording the birth-weight of the infant. The series of mothers was further analysed by race, social status and parity.

The results obtained were clear-cut and impressive. Of 62 infants born at full term from malarious placentae 13 (20·6%) would be classed as "premature", that is, weighing less than 5½ lb. (2,495 gm.), whereas of 378 infants born from uninfected placentae at term, only 31 (8·2%) would be so classified. This difference ($P<0{\cdot}01$) is significant.

Irrespective of the mother's race, the mean birth-weight of children from infected placentae was consistently lower than that of the infants born from uninfected placentae, and this discrepancy is uninfluenced also by differences in social status of the mothers. The difference is however somewhat influenced by parity, since first-born infants tend to have a lower birth-weight than subsequent children whatever the malarial status of the mother.

Compared with Southern and Eastern Nigerian mothers, Northern Nigerian mothers showed in higher proportion the coincidence of parasites in both peripheral blood and placenta. The author considers this a manifestation of their lower degree of immunity, which is to be explained by the strictly seasonal transmission of malaria in Northern Nigeria as compared with the sustained transmission throughout the year which obtains in Southern and Eastern Nigeria.

[It would be of great interest to know the incidence of stillbirths and abortions from malarial and non-malarial placentae, but such an investigation would clearly be very difficult since few would be observed in institutions. This paper records an important, well planned and well executed piece of research.]

[See also this *Bulletin*, 1953, v. 50, 596; 1957, v. 54, 781.]

J. H. Walters

LÓPEZ ANTUÑANO, F. J. Microscopia en la malaria humana. [**Microscopy in Human Malaria**] *CNEP Bol.* 1959, Jan.–Mar., v. 3, No. 1, Suppl., 1–28, 1 fig. & 12 pls.

A fully illustrated description of methods and interpretation of results.

ADAMS, A. R. D. **Drug Treatment of Malaria.** *Brit. Med. J.* 1959, Aug. 15, 183–4, 1 diagram.

For the rational treatment of malaria a knowledge of the various stages of the life cycle of the 4 human species of *Plasmodium* is essential, since the

specific drugs now in use act only at certain points. Details of the parasitic cycle and of the stage at which each individual drug exerts its effect are shown in diagrammatic form.

In recent years the superiority of the 4-aminoquinolines (*e.g.*, chloroquine, amodiaquine) for treatment of the clinical attack has become increasingly recognized. Quinine, however, given intravenously in the form of the dihydrochloride, is the drug most favoured for the treatment of grave or complicated attacks in which speed of action is imperative if life is to be saved.

The 8-aminoquinolines (pamaquin, primaquine) are recommended for radical cure of relapsing *P. vivax*, *ovale* and *malariae* infections.

For prophylaxis of malaria, proguanil and pyrimethamine are recommended. Should local resistance to either of these drugs be encountered, infections may be effectively suppressed by one of the 4-aminoquinolines or by mepacrine. Quinine is not recommended for prophylaxis unless none of the above-mentioned drugs is available.

The appropriate dosage for all the specific drugs mentioned is included in the text. *G. Covell*

SCHRIER, S. L., KELLERMEYER, R. W., CARSON, P. E., ICKES, C. E. & ALVING, A. S. **The Hemolytic Effect of Primaquine. X. Aldolase and Glyceraldehyde-3-Phosphate Dehydrogenase Activity in Primaquine-Sensitive Erythrocytes.** *J. Lab. & Clin. Med.* 1959, Aug., v. 54, No. 2, 232–40, 4 figs. [25 refs.]

BRUCE-CHWATT, L. J. **Assessment of Urinary Excretion of Antimalarial Drugs in Large-Scale Chemotherapeutic Eradication Projects.** *Bull. World Health Organization.* Geneva. 1959, v. 20, No. 5, 973–85. [35 refs.]

Malaria eradication programmes in which mass chemotherapy is used together with the spraying of insecticides are still in the testing stage. Varying degrees of " consumer resistance " are encountered among the peoples to whom the antimalarial drug is distributed ; it is necessary to have some check on the amount of drug taken and the regularity of intake if assessment of the degree of success of each procedure of drug administration is to be made.

One method of doing this is to determine the amount of drug or its metabolites excreted in the urine. To be of practical value in the field, the test must be a simple one. The author surveys the methods which have been used to estimate quinine, mepacrine, chloroquine, proguanil and pyrimethamine, and discusses the advantages and shortcomings of each method. He gives full details of 6 quantitative tests for chloroquine, and draws attention to a method [YUDKIN, this *Bulletin*, 1946, v. 43, 625] in which the approximate daily output of mepacrine in the urine can be calculated from the drug content and specific gravity of a single specimen of urine. Pyrimethamine is used in such low dosage that it is difficult to detect in the urine and present methods of assay are not suitable for field use.

[The methods reviewed are widely scattered in the literature and the author has done a useful service in bringing them together and comparing them. The

Annex to the paper in which detailed descriptions of workable tests for chloroquine are given will be of great value to the field malariologist.]

L. G. Goodwin

GABALDON, A. & GUERRERO, L., with the field collaboration of C. BALESTRINI *et al.* **An Attempt to eradicate Malaria by the Weekly Administration of Pyrimethamine in Areas of Out-of-Doors Transmission in Venezuela.** *Amer. J. Trop. Med. & Hyg.* 1959, July, v. 8, No. 4, 433–9.

The observations here recorded were made in two areas in Venezuela where the malaria eradication programme had failed to achieve complete interruption of transmission. In one of these *Anopheles aquasalis* is the predominant vector, in the other *A. nuñez-tovari*. *Plasmodium vivax* is now practically the only malaria parasite present. Before 1950 most infections produced only a few relapses; now, however, the strain remaining shows a marked tendency to relapse, the inference being that those producing few relapses have been eliminated by the measures adopted.

In both areas spraying with residual insecticides had been supplemented by the distribution of chloroquine through the agency of rural health visitors. In spite of this, fresh infections were encountered from time to time.

During the period July–December 1957 a total of 111,995 persons in the two areas received pyrimethamine once weekly at a dosage of 50 mgm. for adults and correspondingly smaller doses for children. This regime effectively interrupted transmission, as evidenced by failure to find any parasite-positive cases during the last 3 months of the experiment. Relapses appeared in both areas, however, after drug administration had been discontinued. It is hoped that pyrimethamine given at the same dosage, perhaps at fortnightly intervals, will maintain interruption of transmission in such areas until all the infections have died out.

G. Covell

LIVADAS, G. A. **Malaria Eradication in Greece.** *Riv. di Malariologia.* 1958, Dec., v. 37, Nos. 4/6, 173–91. [27 refs.]

The author reviews the programme of malaria control and eradication in Greece, which falls into three periods: 1946–49, of generalized control, 1950–53, discontinuation of spraying and the adoption of surveillance techniques and during which the influence of resistance to insecticides was detected, and 1954–57 when efforts were made to complete the eradication of the disease. A review of recent happenings shows that the discontinuation of spraying for 7 years in Crete had been followed by complete freedom from any single indigenous case of the disease and this island may therefore be considered to be one of the first from which the disease was eradicated. In the remainder of the country, the number of positive cases has remained at a very low level and with only very slight fluctuation. Persistence is of an irregularly distributed pattern, three-quarters of the 1,445 cases identified in 1957 coming from 6 of the 50 prefectures of the country. It is concluded that during the 9 years since general spraying was discontinued there have been no substantial changes in the malaria situation; previous residual foci have not enlarged, some have decreased in size or disappeared and no new ones have arisen to take their place. There is therefore good reason to believe that continuation of these techniques should result in eradication.

G. Macdonald

CAYLA, J. La lutte antipaludique en Turquie. [**The Campaign against Malaria in Turkey**] *Bull. Information Ministère Santé Publique et Population.* Paris. 1958, No. 1/2, 7–13.

Malaria has always been extremely important in Turkey and was indeed one of the main causes of the crumbling of the Ottoman empire, but it had been very greatly reduced though not finally eliminated by control measures carried out with progressive intensity up till 1956. In that year Professor A. CORRADETTI prepared a scheme for eradication of the disease within the next 4 or 5 years. He delimited three main epidemiological zones: (1) the Anatolian plateau, the coast of the Black Sea and the eastern mountains; (2) the coast of the Sea of Marmara and the Aegean Sea; (3) the south-eastern part of the country. The situation was then that the parasite rate among infants had been reduced to 0·10 per 1,000 in region (1), to 0·69 in region (2), and to 3·74 in region (3). It was clear from this and other data that the transmission of the disease had continued on a material scale in this last region.

The scheme of eradication depends primarily on an annual application of a suspension of DDT in all villages below an altitude of 1,500 m. (4,920 ft.) on an administrative background of 1,150 circles, grouped into 248 districts, each intended to be under the control of a medical officer; these in turn are grouped into 34 regions at the headquarters of each of which there are laboratory facilities. Help has been provided in this by UNICEF, but some of the mechanisms were not fully established at the time to which this report takes the reader, 1957.

A review of parasite rates in that year showed that there had been a slight increase in the rate among infants though there had been a further decrease among older children. It is concluded that transmission has continued, particularly in the region of the south-east and that the greatest intensity of effort should be expended in this part of Turkey. With this effort there is good reason to expect success. *G. Macdonald*

KARUNARATNE, W. A. **The Influence of Malaria Control on Vital Statistics in Ceylon.** *J. Trop. Med. & Hyg.* 1959, Apr., v. 62, No. 4, 79–85, 2 maps. [10 refs.]

Ceylon has enjoyed the dual advantages of a very successful malaria control scheme and a very efficient system of registration of vital statistics, from which it has been shown that in the quinquennium in which control was established the crude death rate fell from 20·3 to 12·9 per 1,000, the infant mortality fell from 141 to 82 per 1,000 and the maternal mortality rate from 15·5 to 5·8. The significance of these crude results is however lessened by the fact that they refer to a country in which the influence of malaria had varied very greatly in different areas.

The present study includes the selection of three index areas, each believed to be representative of one of the three main epidemiological types of malaria in Ceylon, hyperendemicity, epidemicity, and evanescence or freedom from the disease. For each of these index areas there are given more detailed statistics, age-specific death rates for pre-control and post-control periods, infant mortality rates for each year from 1944 to 1956 and the sex-specific death rates for the age-group 15–44 years. The death rate in the hyperendemic index area fell in

almost all age-groups to one-third of its previous figure and showed marked but lesser reductions in the other two areas. The infant mortality rate showed a comparable change, from 207 in 1946 to 102 in 1948 in the hyperendemic area, from 124 to 75 in the epidemic area and from 90 to 77 in the area of evanescence. Before control the death rate among women aged 15–45 in the hyperendemic area was double that among men, 2,234 per 100,000 as compared with 1,149; 5 years later the disparity remained but on a greatly reduced scale, 475 as compared with 292; an even greater difference had been apparent in the epidemic zone, 1,928 as compared with 497, but after control the ratio between the two was much as in the hyperendemic area, 357 and 198. In the area of evanescent malaria the distinction was always much less marked, 661 to 501 and 303 to 209.

It is believed that these figures show an unprecedented change in recorded death rates after a public health measure and also demonstrate that the control of malaria has very greatly reduced the mortality among women of child-bearing age, which leads to the conclusion that directly or indirectly malaria had previously greatly increased the risks of maternity.

Changes in the birth rates are also recorded but they are smaller and much more difficult to interpret. *G. Macdonald*

STEYN, J. J., BRINK, C. J. H., BOTHA, H. P., PRETORIUS, H. M., COMBRINK, H. J. & VOSLOO, A. Dragtigheids-data van *Anopheles gambiae* Giles en Malariauitwissing in die Transvaal. [**Biological Data on** *Anopheles gambiae* **in Relation to Malaria Control in the Transvaal**] *South African Med. J.* 1959, Aug. 15, v. 33, No. 33, 679–80.

The English summary appended to the paper is as follows:—

"It would appear that *gambiae* ovulates in 2·2375 days, sits for 4·8818 hours in an unsprayed hut before feeding, and is knocked down in a BHC-sprayed hut in 4·31 hours. It has already been determined that spraying huts with BHC remains effective for 6 months. Malaria eradication by means of spraying huts with BHC is therefore based on a sound rationale."

YEO, D. & WILSON, D. Bagster. **Aircraft Applications for Anopheline Control: Prevention of a Seasonal Increase with a Granulated Formulation of Dieldrin.** *Ann. Trop. Med. & Parasit.* 1958, Dec., v. 52, No. 4, 402–16, 5 figs.

The large number of narrow low-lying valleys containing many small breeding places lying in and around Dar-es-Salaam, Tanganyika, constitute a difficult problem in the control of transmission of malaria. Traditional methods have long been used, and attempts have been made to improve upon them particularly by the experimental use of larvicides applied from the air. Previous experiments [this *Bulletin*, 1947, v. 44, 1039] have been made with DDT solutions which have given imperfect though promising results, their partial failure perhaps being attributable to interception of the droplets by vegetation.

The experiments now recorded and in which it was hoped that these difficulties might be overcome, were carried out with a granulated formulation

containing 2% (w/w) of dieldrin, with a median diameter of just under 0·7 mm. and a mean weight of about 0·1 mgm. applied at the rate of 1 lb. to the acre from a low flying aircraft. The distribution of granules was sampled in boxes roughly corresponding in size to hoofprints and scattered throughout the treated area, from which it was noted that dosages were very variable, but about 90% of these boxes received at least one granule. Entomological evaluation was complicated by the concurrent practice of other methods of mosquito control but a number of catching stations were selected some of which were believed to represent the results of aircraft applications only, others to represent the results of ground work only and others to represent a combination. Spraying was carried out between April and June 1957. The larval searches, which were very thorough, revealed fourth stage larvae on only 7 out of 22 searches between April and August in the places affected by aircraft alone, and the few positive searches were extremely scanty. Large catches were recorded up to mid-July in the stations not affected by control measures. Searches for adult *Anopheles gambiae* in the areas affected by aircraft alone in each of the months from April to December revealed 8·1, 4·1, 0·7, 0·2, 0, 0, 0, 0, 0, females per room, the corresponding figures in stations affected by ground practice alone being 2·4, 2·3, 0·53, 0·08, 0·35, 0·31, 0·34, 0·05 and 0. In untouched stations the figures never sank below 4·3. Contrast with happenings in the previous year suggested a progressively improving reduction in *A. gambiae* populations from 47% in April rising to 100% in August, which was maintained to the end of the year. Reduction in *A. funestus* was comparable though slightly below. The cost of this work was about £100 per sq. mile for each application, which may be compared with a cost of £50 per sq. mile for each application of ground control.

The results are not unequivocal but they do give the most promising indication of a very substantial reduction in breeding at the critical periods of the year. *G. Macdonald*

ADAM, J. P., HAMON, J. & CHEVALIER, J. Observations complémentaires sur la résistance aux insecticides chez les moustiques de la région d'Abidjan (Basse Côte-d'Ivoire). [**Further Observations on Insecticide Resistance in Mosquitoes in the Abidjan Region, Lower Ivory Coast, French West Africa**] *Bull. Soc. Path. Exot.* 1958, July–Aug., v. 51, No. 4, 662–6.

The authors report the results of tests on dieldrin-resistant sources of *Anopheles gambiae* near Abidjan, which is on the lagoon in the south of the Ivory Coast. [Dieldrin-resistant *gambiae* have been reported from many places in West Africa, from Liberia to the Congo; but this appears to be the first published record on the sea coast.] Tests were done on adults (by the WHO method) and on larvae (by the method of ELLIOTT [this *Bulletin*, 1959, v. 56, 418]). On the basis of the genetical work of DAVIDSON [see *ibid.*, 1958, v. 55, 1078] the authors used two discriminating doses to detect the resistant gene in heterozygotes and homozygotes. On the basis of the adult test, there appeared to be 20% heterozygotes and 7% homozygotes at one locality and 11% and 3% respectively, at another. The larval tests gave roughly the same figures for homozygotes. Tests with DDT indicated a very high susceptibility to this insecticide (less than 0·25% LC50) compared with other records for *gambiae*.

Tests on adult *Culex p. fatigans* in the vicinity of Abidjan indicated high resistance to both DDT and dieldrin (low mortality at 4% of either insecticide).

[See also this *Bulletin*, 1959, v. 56, 419.] *J. R. Busvine*

JONES, J. C. **Some Effects of Dieldrin on *Anopheles* Mosquitos.** *Bull. World Health Organization.* Geneva. 1959, v. 20, No. 5, 987–90.

Third and fourth stage larvae and pupae of *Anopheles quadrimaculatus* were tested by continuous exposure to dieldrin, 0·02 to 0·0002 part per million, or to short periods in higher concentrations of 0·5 to 1·0 p.p.m.

All third stage larvae died within 24 hours when continuously exposed to 0·004 p.p.m. of dieldrin and less than 20% of third stage and one-day-old fourth stage survived 0·5 p.p.m. for 15 to 20 minutes. 2-day-old fourth stage were however more tolerant and 40 minutes' exposure to 0·5 p.p.m. was necessary to produce a 90% kill.

5 minutes' exposure to 0·5 p.p.m. of 1- to 18-hour-old pupae killed less than 50% but produced a striking kill during the first 3 days of adulthood. 30 to 50 minutes' exposure resulted in all adults dying by the second day of adulthood. It was also found that male pupae were more sensitive than female pupae and older pupae were more tolerant than younger pupae. *E. R. Nye*

VAN DEN ASSEM, J. **A Window-Trap Hut Experiment to Test the Effects of Dieldrin under Local Conditions in the Merauke Area (Netherlands New Guinea).** *Trop. & Geograph. Med.* Amsterdam. 1959, Mar., v. 11, No. 1, 32–43, 4 figs. [14 refs.]

An account is given of an experiment to test the effect on medically important mosquitoes, in the Merauke area of Netherlands New Guinea, of house applications of dieldrin.

Anophelines (mainly *Anopheles amictus hilli*, *A. bancrofti* and *A. farauti*) entering specially constructed trap huts were subject to the residual effect of dieldrin deposits (500 mgm./sq.m.) on the inside surfaces (lined with brown packing paper). 100% mortality of blood-fed females was observed up to 5½ weeks after spraying. From that time onwards, small numbers survived in every succeeding test but deposits still had some effect up to 5 months after spraying (20% kill). With a 6-month-old deposit no effect could be detected. A "sufficient kill" (assumed to be about 60%) was obtained in a 3-month period after spraying.

Of the other mosquitoes investigated: surviving culicines (blood-fed *Culex annulirostris* and *C. fatigans*) were present in every test from 9 days after spraying; 67% mortality was recorded for blood-fed *Mansonia* spp. on deposits 28 to 46 days old.

Susceptibility tests by means of the WHO adult test kit indicated that there was no resistance to dieldrin in *A. farauti* and *A. bancrofti*. The *Culex* spp. [as might be expected] turned out to be less susceptible.

Although dieldrin is of very real value in malaria control, it is considered impossible to eradicate the vectors of malaria in Dutch New Guinea by indoor dieldrin spraying alone. It is also improbable that the vectors' average

expectation of life is cut down to a point below the length of the extrinsic cycle of the parasite.

An increase in the frequency of spraying, while it would seem advisable, is economically impossible in that country.

Under conditions in New Guinea dieldrin has proved to be inferior to DDT as a residual insecticide.

[See also this *Bulletin*, 1956, v. 53, 154.] *R. J. Wood*

TRYPANOSOMIASIS

In this section abstracts are arranged as far as possible in the following order:—African—*human, animal;* American—*Chagas's disease and other trypanosome infections. In each form the following order is followed:—epidemiology, aetiology, transmission, pathology, diagnosis, clinical findings, treatment, control.*

FRIEDHEIM, E., GEIGY, R. & KAUFFMANN, M. **Drug Responses of the Sakwa Trypanosome (Heisch 1958).** [Miscellanea.] *Acta Tropica.* Basle. 1959, v. 16, No. 2, 165–9.

Recently HEISCH *et al.* [this *Bulletin*, 1959, v. 56, 698] were able to transmit a trypanosome of *brucei*-type from bushbucks to rats and then from rats to man. They designated this parasite *Trypanosoma rhodesiense* and it is usually resistant to tryparsamide but sensitive to Mel B (an arsenical compound in combination with BAL) whereas *T. gambiense* is generally sensitive to both.

It appeared to the present authors worthwhile to test the sensitivity of the freshly isolated trypanosome to both drugs. 107 rats of weight 53 to 64 gm. were infected. There was a slight difference in the number of passages which the strain had undergone in rats before use in the present experiments. 4 groups of 8 rats were treated respectively with 400, 200, 100 and 50 mgm. tryparsamide intraperitoneally per kgm. 4 other groups of 8 rats were similarly treated with 30, 15, 7·5 and 3·75 mgm. per kgm. respectively of Mel B. 2 further groups of 8 rats and a group of 9 were given 800 and 600 mgm. tryparsamide per kgm. followed 4 days later by 15 mgm. Mel B. per kgm. by the same route. Suitable controls were used in each experiment. Results showed that the infection was insensitive to tryparsamide but sensitive to Mel B in agreement with the usual response of *T. rhodesiense* to these drugs [*ibid.*, 1954, v. 51, 152]. *J. D. Fulton*

PACKCHANIAN, A. **On the Cultivation of** *Trypanosoma brucei in vitro.* *Amer. J. Trop. Med. & Hyg.* 1959, Mar., v. 8, No. 2, Pt. 1, 168–74, 3 figs. [16 refs.]

It is generally considered that successful cultivation of pathogenic African trypanosomes was achieved some 30 years ago, since the introduction of Razgha's medium and its modifications [this *Bulletin*, 1930, v. 27, 244; 1938, v. 35, 704]. However, as early as 1904 NOVY and MACNEAL claimed to have

cultivated *Trypanosoma brucei* in the medium (NN) devised by them. Subsequently all attempts to grow this and other pathogenic trypanosomes in a modification of this medium, known as NNN, were unsuccessful, and this medium is usually reserved for cultivation of *T. cruzi* and other trypanosomes of the *lewisi* group. In the meantime the present author has been growing the pathogenic trypanosomes in his own modification of the NN medium [not to be confused with the conventional NNN medium, from which NN differs in essential components].

In this paper a detailed description is given of the composition and preparation of a modification of Novy and MacNeal's original medium, labelled NNP. The medium is prepared in 4 stages, as follows.

(a) *Beef infusion broth*. 500 gm. of finely ground lean beef are thoroughly mixed with 1 litre distilled water; the mixture is allowed to infuse overnight in the cold room (+ 4°C.), and next day it is gradually heated and boiled for 10 minutes, after which the mixture is strained through gauze, and to the hot extract are added 5 gm. NaCl and 10 gm. Difco Bacto-peptone. After adjusting the reaction to *p*H 7·8 with 1/N NaOH, the infusion is boiled for 1 hour, filtered through filter paper until cleared, and its volume is restored to 1 litre with distilled water. Finally the extract is autoclaved in a flask at 15 lb. for 45–60 minutes, and the stock infusion is stored cold (in room or refrigerator).

(b) *Infusion agar*. To 1 litre of above beef infusion broth, after filtration, are added 20 gm. powdered Difco Bacto-agar, which is dissolved by boiling for about 1 hour, after which volume is restored with distilled water, and the mixture is distributed in flasks (100 ml. per 350 ml. flask). This infusion is then autoclaved (45 minutes at 15 lb.) and stored in a cold room.

(c) *Rabbit blood*. 50 ml. of rabbit blood, drawn by heart puncture, are defibrinated in a flask by shaking with glass beads, after which the blood is stored in a cold room for 1–3 days.

(d) *Final medium*. A flask containing infusion agar is melted in a 55°C. water bath, and to the melted agar are added various proportions of rabbit blood, as required, *viz*. 1 to 3 parts (= 75% blood), 1 to 2 (= 66% blood) or 1 to 1 (= 50% blood). The medium is thoroughly mixed and the blood-agar is dispensed in quantities of 6–8 ml. per test-tube; the tubes are slanted with a 10 mm. butt, and after solidification they are stored upright in a cold room.

In this medium the author successfully cultivated 2 strains of *T. brucei*, which were brought from Uganda in 1954, and maintained in Texas by passages in various rodents. Cultures of this trypanosome have been grown in NNP by monthly transfers for periods up to 3 years. All attempts to infect rodents by inoculation of cultures of different age have failed. [From this fact it can be deduced that metacyclic trypanosomes do not develop in this medium.]

Among other species grown in this and similar media are mentioned *T. congolense*, *T. gambiense*, *T. rhodesiense*, as well as species of the *lewisi* group and various other haemoflagellates. *C. A. Hoare*

McDonald, W. A. & Wijers, D. J. B. **Anal Feeding as a Method of infecting Tsetse-Flies with** *Trypanosoma gambiense*. **I.—The Technique.** *Ann. Trop. Med. & Parasit.* 1959, Apr., v. 53, No. 1, 69–71, 4 figs. on pl.

Over a period of 4 years in which laboratory infection with *Trypanosoma gambiense* and other species of the *T. brucei* group was induced in *G. palpalis*,

percentage infection rates in several hundreds of flies, by feeding on infected monkeys and rats, were 0·4% and 0·9% respectively. When day-old flies were used, the rate was raised to 7·6% [this *Bulletin*, 1959, v. 56, 597]. A new approach, that of introducing infected blood to the gut by pipette through the anus, has given 15·8% gland infections of *T. gambiense* in *G. palpalis*. This was in 82 flies dissected 21 days after injection. In 88 control flies (fed on infected monkey) the rate was 5·7%. Flies fed anally, known by probe test to harbour salivary trypanosomes, infected a monkey by bite. Its blood was positive after 11 days. Not only was the trypanosome cycle successful, but, provided the fly's gut was not distended to repletion at anal feeding (this was fatal), survival over 30 days during which further normal blood meals were given [apparently] compared favourably with that of control flies. Survivals of the initial 18–20 flies in each group were 61 and 70% respectively; 12 pupae were obtained from the batch fed anally, and 17 from the controls.

D. S. Bertram

BURSELL, E. **Determination of the Age of Tsetse Puparia by Dissection.** *Proc. Roy. Entom. Soc. of London.* Ser. A. 1959, Apr. 15, v. 34, Pts. 1/3, 23–4.

[See this *Bulletin*, 1934, v. 31, 65.]

NASH, T. A. M. & JORDAN, A. M. **A Guide to the Identification of the West African Species of the *fusca* Group of Tsetse-Flies, by Dissection of the Genitalia.** *Ann. Trop. Med. & Parasit.* 1959, Apr., v. 53, No. 1, 72–88, 12 figs.

Differentiation of tsetse of the *fusca* group—*Glossina fusca*, *G. nigrofusca*, *G. medicorum*, *G. nashi*, *G. tabaniformis* and *G. haningtoni*—in a survey of distribution in British Cameroons was found impossible on external features alone and too slow or uncertain from lateral views of the male hypopygium or from the form of the signum of the uterus in the female, both methods long accepted as adequate. This paper describes and figures diagnostic features as seen in a dorsal view of the male genitalia, and elaborates on the female signum which is subject to considerable variation within species. Methods of dissection are fully detailed. In the males in particular, accuracy of identification is combined with speed.

D. S. Bertram

ESTEVES DE SOUSA, A. **The Use of Phytocides to control Stump and other Secondary Plant Growth in Areas cleared against *Glossina austeni*.** Reprinted from *Bol. Soc. Estudos Moçambique.* 1959, Mar.–Apr., No. 115, 83–108, 3 maps & 7 figs. [13 refs.]

Control of *Glossina austeni* was carried out by forest and thicket clearing with good results in Mozambique. Regeneration from the cut stumps of some forest species was, however, rapid and experiments were carried out to assess the value of phytocides to prevent this regeneration. Solutions of the butyl esters, 2,4-dichlorophenoxyacetic acid and 2,4,5-trichlorophenoxyacetic acid in illuminating paraffin, for foliage spraying, and diesel oil for stump treatment,

were found effective. Spraying should be carried out after clearing and again towards the end of the rainy season to deal with any regeneration.

E. R. Nye

BENOIT, P. L. G. & VAN SANDE, M. Étude des protéines de l'hémolymphe de *Triatoma infestans* et *Rhodnius prolixus* par ultra-microélectrophorèse en gel de gélose. [**Study of Proteins in the Haemolymph of** *Triatoma infestans* **and** *Rhodnius prolixus* **by Means of Ultramicroelectrophoresis on Agar**] *Ann. Soc. Belge de Méd. Trop.* 1959, Apr. 30, v. 39, No. 2, 135–43, 3 figs.

The English summary appended to the paper is as follows:—

"In interpreting these first results it is sufficient merely to state the fact that the haemolymph from a species of *Triatoma*, and that from one of the genus *Rhodnius* show a definitely different morphology on electrophoresis on agar. On the other hand the phenogram of these 2 phylogenetically close species of Reduviids presents analogies which we have not found in the different genera and species of the Ixodidae studied up till now; these will be the subject of later papers."

WARREN, L. G. & BORSOS, T. **Studies on Immune Factors occurring in Sera of Chickens against the Crithidia Stage of** *Trypanosoma cruzi*. *J. Immunology*. 1959, June, v. 82, No. 6, 585–90. [24 refs.]

Earlier experiments by WARREN [this *Bulletin*, 1958, v. 55, 742] showed that crithidial forms of *Trypanosoma cruzi* growing in chicken macrophage cultures in the presence of chicken serum were rapidly lysed, but were agglutinated when the serum was inactivated. The differences in susceptibility of these forms and of trypanosome stages of *T. cruzi* to chicken serum is noteworthy. The present investigation is concerned with the nature of the immune bodies in normal chickens and those reared under germ-free conditions. A Brazilian strain of *T. cruzi* was used and after the parasites were harvested from 5–10-day cultures by centrifugation they were then washed three times in phosphate saline. The experiments were carried out at 25–28°C. Examinations for agglutination or lysis were made on a slide viewed under low power of the microscope with 2-fold dilutions of sera, which, when required were inactivated at 56° for 30 minutes.

Lysis appeared to require the presence of a heat stable factor plus another which was heat labile. Agglutinating factors appeared in the sera of embryos on the 15th day of incubation and lytic factors on the 18th day. The agglutinating factor in sera of normal chickens sometimes disappeared after 28 days. In the case of germ-free birds the serum showed a gradual loss of the agglutinating factor and a rise in lytic factor. It appeared from these and earlier results that the lytic factor was in the nature of complement and the agglutinating factor of sensitizing character.

J. D. Fulton

LEISHMANIASIS

In this section abstracts are arranged as far as possible in the following order:—visceral, cutaneous, muco-cutaneous.

FORATTINI, O. P., JUAREZ, E., BERNARDI, L. & DAUER, C. Leishmaniose tegumentar, no território do Amapá, Brasil. [**Cutaneous Leishmaniasis in the Territory of Amapa, Brazil**] *Rev. Inst. Med. Trop. São Paulo.* 1959, May–June, v. 1, No. 1, 11–17, 13 figs. [10 refs.] English summary.

Up to the present there was little information about the occurrence of cutaneous leishmaniasis in the Amazon region of Brazil, though the form called *pian bois* is well known in the neighbouring French Guiana.

The authors record the recent finding of 66 human cases of this disease among miners and agricultural workers living in the vicinity of the forest in the Territory of Amapa. The lesions appear in the form of cutaneous ulcerating sores, without any mucocutaneous involvement, chiefly on the arms and legs, and less frequently on the body. The diagnosis was confirmed by finding leishmaniae in the lesions and by positive Montenegro reaction. In the region in question several species of sandflies are present, and, although none has yet been incriminated as a vector of the disease, it is significant that *Phlebotomus squamiventris* fed readily on human baits.

Illustrations are given of the terrain in the endemic area and of some patients showing the lesions. *C. A. Hoare*

FEVERS OF THE TYPHUS GROUP

In this section abstracts are arranged as far as possible in the following order:—general; louse-borne typhus, flea-borne typhus, mite-borne typhus; rickettsialpox; tick-borne typhus; Q fever, other rickettsial diseases.

JUMINER, B., with the technical collaboration of A. ZAIBI & D. GHARBI. *Rattus norvegicus* et rickettsioses. Rôle du rat en tant que réservoir de virus probable du typhus épidémique et de la fièvre Q. [**Role of *Rattus norvegicus* as a Probable Reservoir of Epidemic Typhus and Q Fever Rickettsiae**] *Arch. Inst. Pasteur de Tunis.* 1959, June, v. 36, No. 2, 173–82, 2 graphs & 3 figs. on 2 pls.

Micro-agglutination tests with rickettsial antigens on sera from 400 *Rattus norvegicus* collected in Tunis during the period November 1958–March 1959 gave positive results at the dilutions indicated; with Q fever antigen, 9 positives (2·2%) at 1 in 20; with murine typhus antigen, 13 (3·2%) at 1 in 160; with epidemic typhus antigen, 80 (20%) at 1 in 320. No positive results were obtained with boutonneuse fever antigen (sera diluted 1 in 160). The frequency of positive results with epidemic typhus antigen was notably high in November, December and January, falling sharply in February and March. At the dilutions mentioned above, only 3 sera agglutinated more than 1 antigen; these 3 sera agglutinated both epidemic and murine antigens.

These results are taken as an indication that *R. norvegicus* may be a reservoir of *Rickettsia prowazeki* and *R. burneti*, as well as *R. mooseri*, in Tunis. Work aiming at the isolation and identification of rickettsiae from the brains of rats is in progress; the possibility that there may be a natural cycle of transmission

of rickettsiae other than *R. mooseri* which involves rats and men will also be examined.

R. S. F. Hennessey

NICOLESCO, I., POPA, S., BĂLTEANU, Ecaterina & SOLOMON, Hedwiga. Importance de la réaction de fixation du complément dans l'étude d'une épidémie de typhus exanthématique. [**The Importance of the Complement-Fixation Reaction in the Study of a Typhus Fever Epidemic**] *Arch. Roumaines Path. Exper. et Microbiol.* 1959, Mar., v. 18, No. 1, 89–98. [31 refs.]

LEVY, B. **Scrub Typhus in the Differential Diagnosis of Venereal Disease.** *J. Roy. Army Med. Corps.* 1959, July, v. 105, No. 3, 125–7.

"Two cases of scrub typhus are described. In both the existence of a genital eschar with regional adenitis pointed to a venereal infection. Although only two cases have been recorded during the previous two years at the Venereal Disease Department of the British Military Hospital, Singapore, it is possible that others have been missed and it is considered that scrub typhus should enter into the differential diagnosis of certain types of genital ulcers, particularly those occurring in soldiers serving in areas where scrub typhus is endemic."

See also p. 1289, HEISCH & MCPHEE, **Notes on Some Ixodid Ticks in the Nairobi Area.**

MARMION, B. P. **Q Fever: Recent Developments and some Unsolved Problems.** [Abridged.] *Proc. Roy. Soc. Med.* 1959, Aug., v. 52, No. 8, 613–16 (Sect. Epidem. & Preventive Med. 25–8), 1 fig. [24 refs.]

ANGELILLO, B., CORTICELLI, B. & MASIA, G. F. La ricerca degli anticorpi contro *C. burneti* nei bovini mediante l'impiego della reazione di conglutinazione (C.C.A.T.). [**The Search for Antibodies against** *C. burneti* **in Bovines by the Conglutination Reaction (CCAT)**] *Igiene Moderna.* 1959, v. 52, Nos. 3/4, 167–74. [12 refs.]

The English summary appended to the paper is as follows:—

"The search for antibodies against *C. burneti* has been comparatively carried out in 240 bovine sera, by the complement fixation and the conglutination reaction. Results were found to correspond in 92·07 per cent of the cases. In positive sera, the titer obtained by the conglutination reaction was generally higher than that yielded by the complement fixation."

[See this *Bulletin*, 1949, v. 46, 456.]

ZARNEA, G., ALEXANDRESCU, N., SZEGLI, Lucia, VOICULESCU, R., DINCULESCU, M., IONESCU, Honorina & PEREDERI, S. **Occurrence of** *Coxiella burneti* **in a Hedgehog (***Erinaceus europaeus* **[Linné]) captured in an Endemic-Enzootic Area of "Q" Fever.** *Arch. Roumaines Path. Expér. et Microbiol.* 1959, Mar., v. 18, No. 1, 83–8, 3 figs.

The English summary appended to the paper is as follows:—

"Investigations carried out in an endemo-enzootic area of 'Q' fever in an agro-zootechnical farm, revealed the presence of Coxiella burneti in the hedgehog Erinaceus europaeus (Linné). This was proved by the presence of positive

serological reactions (CFT) and the isolation of the etiological agent from the spleen and adrenal glands of one of the captured animals.

"Considering the biology of the hedgehog, its direct epizootic implication as a source of infection to other animal species seems very reduced; its infection proves however, that 'Q' fever has spread to the animals on which the hedgehog feeds: insects, small rodents, etc.

"The presence of infection in the hedgehog was secondary to the focus of 'Q' fever which developed in the farm, where the chief source of infection were the domestic animals from which the disease spread to wild animals and to man."

YELLOW FEVER

In this section abstracts are arranged as far as possible in the following order:—epidemiology, aetiology, transmission, pathology, diagnosis, clinical findings, treatment, control.

CAUSEY, O. R. & MAROJA, O. **Isolation of Yellow Fever Virus from Man and Mosquitoes in the Amazon Region of Brazil.** *Amer. J. Trop. Med. & Hyg.* 1959, May, v. 8, No. 3, 368–71.

That yellow fever is always present in the Amazon Valley has been shown by records of fatal cases in every year except 6 between 1935 and 1957. Two small epidemics in 1954–55 are described. The first was detected by close surveillance of labourers engaged in clearing virgin rain forest in the Oriboca Forest near Belém. The first 3 cases occurred about a week after work started and a further 6 cases followed during the subsequent month. During the following 3 months 110 labourers were examined but no further cases were detected. At the end of this period the labour force was vaccinated but new labour was later introduced and a further case occurred. Severe headache and epigastric pain were the commonest symptoms and a low to normal leucocyte count was the rule early in the disease. Only 3 of the patients had mild attacks: none died. Viruses subsequently identified as yellow fever were isolated from the blood of all of them and all had a significant neutralizing antibody response. Viruses were also isolated from 4 pools of mosquitoes caught in the forest during the epidemic (2 pools of *Haemagogus*, 1 of sabethine, and 1 of *Aëdes* + sabethine mosquitoes).

The second outbreak was among forest-dwellers who cultivate clearings and cut for charcoal in the Apeu Forest. 11 strains of yellow fever virus were isolated from 105 sick persons bled. In all, 36 of 71 patients examined serologically yielded virus or showed antibody. There was one fatality. The outbreak lasted for about a month, being terminated by mass vaccination.

C. E. Gordon Smith

COLLIER, W. A., DE ROEVER-BONNET, H. & HOEKSTRA, J. **Changed Virulence of the Yellow Fever Virus Vaccine Strain 17D by a Single Mouse Passage.** *Trop. & Geograph. Med.* Amsterdam. 1959, Mar., v. 11, No. 1, 75–9, 5 graphs.

17D vaccine was inoculated intracerebrally into mice at a series of dilutions from 1 in 256 to 1 in 262,144 and the brains of paralysed mice from each

dilution were titrated by intracerebral inoculation. The brains were ground in 1% bovine albumin in saline [apparently unbuffered]. The titre of the vaccine was $10^{-4.5}$ and the titres of the passaged brains varied from 10° to $10^{-5.5}$

The " virulence " of these preparations is compared by plotting the cumulative titres on each day of the experiment (by the Reed-Muench method, *Amer. J. Hyg.*, 1938, v. 27, 493) against time. [These curves appear to have only a limited value in the absence of estimates of their confidence limits.]

[See also this *Bulletin*, 1955, v. 52, 1185.] *C. E. Gordon Smith*

Collier, W. A., De Roever-Bonnet, H. & Hoekstra, J. **A Neurotropic Variety of the Vaccine Strain 17D.** *Trop. & Geograph. Med.* Amsterdam. 1959, Mar., v. 11, No. 1, 80–92, 4 graphs.

17D vaccine was passaged intracerebrally in mice. The mean survival period of the mice shortened from 10·7 days in the first and 11·2 in the second passage to an average of 7 days in the eleventh-twentieth passages, and to an average of 6·5 days between the 47th and 65th passages. The " virulence " is compared as in the previous paper [this *Bulletin*, 1959, v. 56, 714] and maximum virulence appears to be reached by the twelfth mouse passage. The mouse-adapted 17D appears to have a virulence intermediate between 17D and neurotropic Asibi virus.

Cross-challenge experiments demonstrated (a) that 17D given intraperitoneally protected against 17D intracerebrally, but not against Asibi and only after several doses against mouse-adapted 17D ; (b) that mouse-adapted 17D given intraperitoneally protected against both 17D and mouse-adapted 17D intracerebrally, but not against Asibi ; (c) that Asibi given intraperitoneally protected against all 3 strains given intracerebrally ; (d) that 17D given intracerebrally (in doses too small to affect the mice) protects against mouse-adapted 17D given intracerebrally. These experiments suggest that there may be antigenic differences between 17D, mouse-adapted 17D, and mouse-adapted Asibi yellow fever viruses, although they have a common origin in viscerotropic Asibi virus. This requires further investigation. [Proof of antigenic changes in arborviruses due to changes of host would have important implications.] *C. E. Gordon Smith*

RABIES

Edge, G. A. **The Challenge of Rabies.** *Canadian J. Pub. Health.* 1959, Aug., v. 50, No. 8, 309–12.

In this article the author refers to the recent southward spread of rabies from the North West Territories, where the disease has been long enzootic among the wildlife population, to the older, more populated areas of Canada south of the 60th parallel, and deals specifically with the problem thereby occasioned in Ontario, one of the Provinces mainly affected. Despite its extensive land frontier with the United States of America, where rabies has been enzootic for

over 60 years, Canada had until the 1950s remained remarkably free from that disease. Thus between 1906, when the first 2 outbreaks (of canine origin) occurred, and 1952, fewer than 1,000 cases of animal rabies were reported for the whole of Canada. In the 5-year period 1953–57, however, another 1,000 cases were reported and during the year 1958 Ontario alone reported 2,485 cases—an increased incidence attributable to the southward and eastward spread across Canada in the 1950s of rabies in foxes. The gravity of the situation in Ontario was reflected in the progressive increase in incidence throughout 1958, from 70 cases reported in January from 12 counties or districts to a peak in December of 640 cases spread over 33 counties.

A study of the distribution by groupings of animals—household, farm and wild—showed that household pets—dogs and cats—accounted for 6·1% of the total cases, farm animals, principally cattle, for 44·2% and wild animals, principally the red fox, for 49·7% ; that the number of affected skunks seemed to be increasing (approximately 3% of all reported cases) ; and that to date no confirmed cases had occurred among small ground rodents or among bats.

Inasmuch as rabies is enzootic in Ontario among wild animals, principally the fox, and as such animals do not lend themselves to quarantine, vaccination or tie-up regulations, other measures to contain the infection and reduce its spread must be sought ; such measures—the reduction in number of wildlife species, the vaccination of farm and household animals, and the education of the public as regards the application of these measures to affected areas—might well achieve the desired result. In this connexion it is suggested that the public health authority might profitably serve as a coordinator of the various authorities involved in the prevention of rabies.

[The course of known outbreaks of rabies in Canada in the 20th century was outlined and the current extensive enzootic described by PLUMMER (*Bull. World Health Organization*, 1954, v. 10, 767 ; this *Bulletin*, 1954, v. 51, 1238).]

G. Stuart

KHEMANI, K. M. & JHALA, H. I. **Study of Rabies Virus Tropism for Salivary Glands.** *Indian J. Med. Sci.* 1959, May, v. 13, No. 5, 420–22.

The brains and submaxillary salivary glands of 145 dogs suspected of rabies were examined in the Haffkine Institute, Bombay. Brains without Negri bodies were tested for virus by inoculation into mice. All the salivary glands were inoculated into mice. The brains of fatally affected mice were examined for Negri bodies. Evidence of rabies infection was found in 97 brains but only 66 (68%) of the corresponding salivary glands had virus isolated from them.

C. E. Gordon Smith

PLACIDI, L. Note sur l'incubation prolongée ou la latence du virus rabique. Cas de la souche Flury. Observation et discussion. [**Prolonged Incubation Period, or Latency of Rabies Virus. Study of the Flury Strain. Observation and Discussion**] *Ann. Inst. Pasteur.* 1959, Aug., v. 97, No. 2, 265–7.

The brains of 4 hedgehogs dying after intracerebral inoculation with Flury virus [this *Bulletin*, 1958, v. 55, 498] were ground up and each inoculated intracerebrally into hedgehogs, guineapigs and rabbits. The guineapigs and rabbits

inoculated from 2 of the 4 hedgehogs had very long incubation periods: the guineapigs died 229–256 days after inoculation and 2 of the rabbits after 242 and 400 days; 2 rabbits survived.

The author states that "it is not always possible to isolate the virus from the central nervous system of these animals". [But no evidence is given that these animals died of rabies infection.] *C. E. Gordon Smith*

MÖBEST, H. Über die Schutzwirkung des Hempt-Impstoffes gegen Tollwut bei verkürzter Immunisierung und vorverlegter Nachinjektion. [**The Protective Effect of Hempt Vaccine against Rabies with Abbreviated Immunization and Early Booster Injection**] *Arch. f. Hyg. u. Bakt.* 1959, Feb., v. 143, No. 2, 135–9.

By means of the Habel test an investigation has been made of the number of doses of Hempt rabies vaccine which are required to produce immunity in mice. In preliminary tests it was found that the same degree of immunity was obtained with vaccine diluted 1 in 10 or 1 in 20. In the main investigation individual groups of mice were given 1–6 injections of 3 batches of Hempt vaccine and 1 batch of Semple vaccine and challenged on the 15th–16th day after the first injection. The protection indices observed after 1 or 2 doses of vaccine did not exceed 1·09 log unit, whereas after 3–6 doses values of 1·36–2·55 log units were observed. The optimum timing of a booster injection is also discussed. *D. J. Bauer*

NICOLAU, S. S., DRAGANESCO, N., NICOLAU, C. S., FUHRER, B., GIRD, E. & IONESCO, N. I. **A New Type of Anti-Rabies Vaccine; Investigations into the Migration of the Vaccine Virus in the Animal Organism by means of Radioactive Substances.** *Acta Virologica.* Prague. 1959, v. 3, Suppl., 91–6, 1 fig. [16 refs.]

The new type of vaccine used in the first series of experiments detailed in the present paper was prepared from a 5% suspension of rabbit brain infected with fixed rabies virus and attenuated by a 0·15% solution of silver nitrate. This vaccine, which on intracerebral inoculation proved to be wholly innocuous for mice, was found to confer on mice and rabbits a degree of protection (against subsequent intracerebral challenge with fresh fixed virus) comparable with that afforded by "the best attenuated anti-rabies vaccines known".

In the second series of experiments the authors, by substituting radioactive $^{110}AgNO_3$ for ordinary $AgNO_3$ in the preparation of their "silver" vaccine, sought to follow the distribution and accumulation of the vaccine in various tissues of immunized rabbits. 2 groups, of 6 rabbits each, were injected in the thigh muscles with 2 ml. doses of the radioactive vaccine and the control suspensions (healthy rabbit brain treated with $^{110}AgNO_3$), respectively, while a third group (3 animals) served to control the activity of the virus used for vaccine preparation. In the third group all 3 animals died shortly after intracerebral inoculation of 0·3 ml. of a 10% fixed rabies virus suspension. 5 determinations of radioactivity were made with each of 14 tissues taken from the 12 experimental animals (in groups 1 and 2) killed at intervals of 1 to 16 days

after injection. These tissues included sciatic nerves, various parts of brain and spinal cord, paravertebral muscles, kidneys, liver, spleen, adrenals, bone-marrow and mesenteric ganglia.

Results showed, *inter alia*, that, although ^{110}Ag could not be found in any of the tissues examined 24 hours after injection of either vaccine or control brain suspension, it could be found in varying concentrations in different tissues 48 hours thereafter; that the sciatic nerve of the inoculated thigh (the homolateral) was the only tissue in which ^{110}Ag was detected regularly; that in other tissues it was present irregularly, although found in almost every case in the contralateral sciatic; that the amount of ^{110}Ag in the sciatic of animals inoculated with radioactive vaccine was much higher than in all the other tissues; and that on the 16th day after inoculation the homolateral sciatic nerve of animals inoculated with radioactive vaccine contained about 7 times as much radioactivity as the amount found in animals inoculated with radioactive control brain material.

Inasmuch as the radioactivity of the homolateral sciatic nerve was thus shown to be higher than that of any of the 14 different tissues studied, the neurotropic affinity of the attenuated virus contained in the vaccine was clearly demonstrated, although the vaccine was not pathogenic for mice on intracerebral inoculation. *G. Stuart*

YURKOVSKY, A. M. & GHENDON, Y. Z. **A Study on the Content of Neutralizing Antibodies in Protein Fractions of Hyperimmune Horse and Bovine Rabies Antisera.** *Acta Virologica.* Prague. 1959, July, v. 3, No. 3, 153–8. [14 refs.]

" 1. Specific anti-rabies antibodies were found in beta, T and gamma globulin fractions of hyperimmune horse and bovine rabies antisera.

" 2. For the preparation of purified and concentrated horse or bovine rabies antisera, methods of purification and concentration are necessary which ensure concentration of the whole complex of beta, T and gamma globulins.

" 3. Albumins as a ballast fraction have to be fully removed."

HOSTY, T. S., KISSLING, R. E., SCHAEFFER, M., WALLACE, G. A. & DIBBLE, E. H. **Human Antirabies Gamma Globulin.** *Bull. World Health Organization.* Geneva. 1959, v. 20, No. 6, 1111–19.

To obviate the risk of foreign protein reactions occasionally experienced with the use of equine hyperimmune anti-rabies serum in the specific post-exposure treatment of man, an attempt was made to prepare a rabies antiserum of human origin. To this end a killed rabies virus duck-egg vaccine was employed for the primary course of immunization, 1 ml. being administered subcutaneously in the scapular region to each of 34 volunteers at 4-day intervals until a total of 5 injections had been given. 30 days after the last injection, *i.e.* on the 46th day, 0·2 ml. of Flury HEP (high egg passage) chick-embryo vaccine was given intracutaneously to each participant and this was followed by a final 1 ml. dose of duck-egg vaccine on the 288th day. 24 days after the last booster dose, *i.e.* on the 312th day, 500 ml. of blood were drawn from each participant

showing significant antibody levels, the serum being pooled and converted to gamma globulin. The pooled serum, taken from people having no pre-vaccination antibody and responding with variable titres to the immunization schedule, proved, on subjection to the "regular" neutralization test described by ATANASIU *et al.* [this *Bulletin*, 1958, v. 55, 754], to have a titre of 1 in 100 against 50 LD50 doses of rabies virus and the gamma globulin prepared from this pool a titre of 1 in 300. By contrast a refined serum from hyperimmunized horses showed a protective titre of 1 in 5,164 against 239 LD50 of fixed virus.

The results of antibody determinations by the "modified" neutralization test of Atanasiu *et al.* [*loc. cit.*], in the sera of 5 volunteers who had shown no rabies neutralizing antibody in a preliminary test and who had been inoculated intramuscularly with the human rabies immune gamma globulin (approximately 8% solution) in a dosage of 0·5 ml. per kgm. of body weight, are then given and compared with those obtained in the sera of 3 persons inoculated with the above-mentioned hyperimmune horse serum in identical dosage. 4 of the 5 persons treated with human gamma globulin had significant titres (a neutralization index of 0·50 or more) on the 15th day and all 3 of those given horse serum had a significant antibody level at that time. The same ratio persisted on the 21st day, but the decrease in titre was more gradual with the human globulin.

From the results of the present study the authors tentatively conclude that gamma globulin of human origin may serve to provide a treated person with immediate passive antibody with which to inactivate virus introduced during exposure and possibly protect for a long enough period until active immunization is induced by the vaccine recommended to be given simultaneously.

Finally it is expected that higher titres in the gamma globulin could be obtained if persons were inoculated with more potent vaccines and if, subsequently, only those responding with high titres were selected for the pooling of serum before its conversion to gamma globulin. *G. Stuart*

CHOLERA

In this section abstracts are arranged as far as possible in the following order:—epidemiology, aetiology, pathology, diagnosis, clinical findings, treatment, control.

CHIBRIKOVA, E. V., KUZNETSOVA, V. I., BAZUNOVA, L. P. & DUDKOVA, V. K. **The Accelerated Detection of *Vibrio comma* in Water and in Washings from Objects in the Surroundings by means of Fluorescence Microscopy.** *J. Microbiol., Epidemiol. & Immunobiol.* London. 1958, v. 29, No. 10, 1591–5. [Translated from Russian.]

This paper should be read by all those who are likely to be concerned with the isolation of *Vibrio cholerae*.

The method devised by the authors is that a water sample or an aqueous washing of material being examined bacteriologically for *V. cholerae* is filtered

through membrane filters in the usual way. In order to grow the bacteria held on them, the filters are then laid on solid differential medium with sucrose which promotes the growth of *V. cholerae*; incubation is at 37°C. for 8–9 hours. After this the filters with the miniature colonies which have developed on them are treated with a solution of fluorescing dye and examined under a binocular microscope, with illumination from above through a pale blue filter to make the colonies fluorescent. Colonies resembling *V. cholerae* are taken from the surface of the medium with a fine capillary pipette and are transferred to a series of tubes for testing with specific phage, cholera agglutinating O antiserum diluted in broth, and for other characteristics. These tubes are incubated at 37°C. for 2½–3½ hours and assessment of the relationship of the isolated bacteria to *V. cholerae* made on the basis of the following data: morphology of the colonies on the membrane filter, typical morphology and motility of cells, ability to be lysed by specific phage, positive agglutination reaction with O antiserum to full titre, and ability to ferment starch.

Thus, 11–12 hours after inoculation of the material under investigation a final conclusion can be made but a tentative answer can be given earlier, namely 8–9 hours after the beginning of the investigation, on the basis of the morphology of the young colonies on the filter, cell morphology, and a positive micro-agglutination reaction of the suspected colonies with cholera O antiserum.

The method has been used for detecting *V. cholerae* in artificially infected water, variously polluted, and a suspension of *V. cholerae* at a concentration of 10 cells per ml. added to these water samples. In almost all cases it was possible to isolate a culture of *V. cholerae* and give a final answer 11–12 hours after beginning the examination. The total number of bacteria of all types in the samples studied was considerable and varied from several hundred to 10,000 per ml.

It was found that membrane filters No. 3 and 4 were suitable for removing *V. cholerae* from water and the preparation of these together with the apparatus was carried out in the conventional manner. Volumes of 5, 10 and 25 ml. of water sample were used and at the end of filtration the filters were washed lightly with 1% peptone water (*p*H 7·8) and then transferred to plates of nutrient medium.

Careful consideration was given to the constituents in the medium and that finally selected had the composition:—1% Hottinger agar of *p*H 7·8, containing 1·50 mgm. % of amino-nitrogen (base) + 1·5% sucrose + 0·05% vitamin B1 + 0·3 – 0·5 ml. (to the point of decoloration) of a 10% aqueous solution of sodium sulphite (amount per 100 ml. of medium).

The presence of a considerable amount of amino-nitrogen and vitamin B1 with a reduced agar content ensured the accelerated growth of colonies of *V. cholerae*. The addition of sucrose made possible the differentiation of the colonies on the filter and with the indicator fuchsin in the medium the sucrose fermenting colonies were coloured red and this colour remained after fluorescent treatment of the filters. In fact, the presence of this dye in the medium made for brighter fluorescence of the colonies when they were observed in blue light.

E. Windle Taylor

AMOEBIASIS AND INTESTINAL PROTOZOAL INFECTIONS

In this section abstracts are arranged as far as possible in the following order:—epidemiology, aetiology, pathology, diagnosis, clinical findings, treatment, control.

OSADA, M. **Electron-Microscopic Studies on Protozoa. I. Fine Structure of** *Entamoeba histolytica*. *Keio J. of Med.* Tokyo. 1959, June, v. 8, No. 2, 99–108, 14 figs. on 4 pls.

A brief description, illustrated by photomicrographs, is given of the cytology of *Entamoeba histolytica* in sections of amoebae and cysts from cultures fixed in 1% osmium tetroxide and embedded in a mixture of 3 parts methyl methacrylate and 7 parts n-butyl methacrylate. It was found that the trophozoite is invested in an outer membrane, but there is no difference in the structure of the endo- and ectoplasm, except for the presence of vacuoles in the former. The nuclear membrane consists of two layers with minute perforations, and is lined with chromatin granules which have a high electron-density. Encystation of the amoebae proceeds as follows: the body rounds off, the number of vacuoles decreases until they disappear, then glycogen vacuoles—followed by chromatoid bodies—are produced, and finally the cyst wall is formed. *C. A. Hoare*

FREEDMAN, L. & ELSDON-DEW, R. **Size as a Criterion of Species in the Human Intestinal Amebae.** *Amer. J. Trop. Med. & Hyg.* 1959, May, v. 8, No. 3, 327–30, 6 figs.

In this paper an account is given of experiments designed to demonstrate variation in the size of various populations of *Entamoeba histolytica* in culture and to determine the factors influencing this variation.

It was shown that in 2 lines of amoebae of the same strain, grown with different bacterial associates, the size distribution was bimodal, but when they were mixed and cultivated together the curve became unimodal. In this case it was evident that the difference in the size of amoebae in the 2 lines was due to environmental factors, *viz.* to the kind of bacteria with which they were associated.

In other cases it was shown that the difference in size observed in populations of this amoeba represented genetically distinct groups. Thus, in one case amoebae isolated from a patient fell into 2 groups, the mean measurements of which were $7{\cdot}5\mu$ and $14{\cdot}5\mu$ respectively, and the size distributions of which remained bimodal during the entire period of cultivation of 2 months. In a number of other cases pairs of populations of amoebae, differing in size and in associated floras, also showed a bimodal distribution after growing together in a mixed culture. In another experiment a culture of *E. coli* was grown with bacteria from a culture of *E. histolytica* and *vice versa*. In this case the two species could be differentiated by their size distributions.

In discussing these findings it is emphasized that random measurements of *E. histolytica* provide no clue to the nature of variation in this species. This may be induced environmentally, as in some of the populations studied in this work, or they may be genetically fixed, as in the case of the large and small races. In the latter case the persistence of a bimodal curve provides a good criterion for the differentiation of *E. histolytica* proper from *E. hartmanni*.

Thus, a mean size of 10μ for cysts and 12μ for trophozoites can serve as the dividing line for the separation of the larger species from the smaller one.

[See also this *Bulletin*, 1958, v. 55, 652.] *C. A. Hoare*

FAUST, E. C. & READ, T. R. **Parasitologic Surveys in Cali, Departamento del Valle, Colombia. V. Capacity of *Entamoeba histolytica* of Human Origin to utilize Different Types of Starches in its Metabolism.** *Amer. J. Trop. Med. & Hyg.* 1959, May, v. 8, No. 3, 293–303, 2 pls. [25 refs.]

This paper is devoted to a study of the utilization by *Entamoeba histolytica* of various starches from foodstuffs which form the staple diet of the poorer classes of population in Cali, Colombia. In this connexion it is noted that 40% of the population harbour this parasite without manifesting clinical amoebiasis and pass in their stools undigested starch grains which might presumably be ingested by the amoeba in the large bowel.

In vitro tests were carried out with a local strain of *E. histolytica* grown in Balamuth's medium with rice powder. In the course of subcultures the latter was replaced by various starches obtained from local foodstuffs. It was found that the amoebae grew readily in cultures containing starch from plantain, Irish potato, sweet potato, yucca and wheat, but did not thrive on maize starch. One non-Colombian strain of the amoeba was also grown satisfactorily with substituted starches (yucca and potato), but another failed to establish itself *in vitro*.

The results of these experiments are discussed in the light of available data regarding the nutrition of the local population and the physiology of *E. histolytica*. Thus, the presence of undigested starch particles in the stools is attributed to a protein-deficient diet which is responsible for inadequate enzyme production, such as amylase and maltase. On the other hand, *E. histolytica* elaborates these enzymes, thereby converting the ingested starch granules into sugars, the degree of their utilization depending on whether amylopectin or amylose are preponderant in them: thus, rice and Irish potato starches contain chiefly the former component, wheat and sweet potato the latter, while plantain and yucca occupy an intermediate position.

On the basis of these facts, the behaviour of *E. histolytica* in human infections observed in Cali is thought to be as follows: (a) protein malnutrition causes pancreatic and intestinal enzyme deficiency, on account of which ingested starches are not fully hydrolysed and are thus available to the amoebae in the intestine; (b) since these starches fully satisfy the nutritional needs of the amoebae, invasion of the intestinal wall becomes unnecessary; (c) this fact explains the symptomless course of amoebic infection in the local population, in spite of the pathogenicity of the strains of *E. histolytica* in this geographical area.

A detailed description is given of the various forms of starch tested, and their appearance is illustrated in a series of figures. *C. A. Hoare*

POWELL, S. J. **The Hematological Findings, Serum Protein Pattern and Liver Function Tests in Acute Amebic Dysentery and Amebic Liver Abscess.** *Amer. J. Trop. Med. & Hyg.* 1959, May, v. 8, No. 3, 331–6. [32 refs.]

Published reports on liver function tests in hepatic amoebiasis are conflicting. In this study from Durban, 49 male Africans suffering from acute amoebic

colitis, 31 from indisputable amoebic liver abscesses, and 50 healthy male Africans, labourers from the same environment, were subjected to a series of blood examinations, liver function tests, serum protein estimations and electrophoretic protein determinations. The results are set out in tables. In the cases of amoebic liver abscess the haemoglobin value was low and the erythrocyte sedimentation rate high; most of these had a normochromic anaemia. Leucocytosis (mean 13,000 cmm.) was usual in cases of acute amoebic dysentery as well as of amoebic liver abscess.

In acute dysentery the total serum proteins were significantly lowered, owing to a reduction in the serum albumin; in amoebic liver abscess the total proteins were normal despite a fall in serum albumin; this was due to an increase in the serum globulin. In both conditions the alpha globulin fraction was raised, especially in patients with amoebic liver abscesses, and the beta globulin fractions were somewhat reduced; the gamma globulin fraction was not significantly altered by the dysenteric disease, but it was markedly increased by the liver disease.

The mean alkaline phosphatase and zinc turbidity were increased in cases of amoebic liver abscess; the cephalin cholesterol test was erratic and the serum bilirubin normal in all cases.

In cases of liver abscess the serum albumin and the alkaline phosphatase became normal within a month of successful treatment and the amount of gamma globulin and the zinc turbidity after 3 months.

The findings in cases of acute amoebic dysentery are compatible with bowel dysfunction, and the findings in amoebic liver abscess are indistinguishable from those of hepatic dysfunction. *A. R. D. Adams*

POWELL, S. J. **The Serum Protein Pattern, Liver Function Tests and Hematological Findings in the Differential Diagnosis of Amebic Liver Abscess.** *Amer. J. Trop. Med. & Hyg.* 1959, May, v. 8, No. 3, 337–41. [28 refs.]

The differential diagnosis of amoebic liver abscess from such conditions as primary carcinoma of the liver, hepatic cirrhosis and right basal pneumonia is primarily clinical, but laboratory investigations may at times give some help in establishing it. After reference to his own results, details of which are set out in tables, and the work of others the author, from Durban, reaches the following conclusions:

"1. Leukocytosis is uncommon in cirrhosis.

"2. Elevation of the serum bilirubin is uncommon in liver abscess.

"3. The alkaline phosphatase tends to be highest and most consistently raised in primary carcinoma. It is rarely elevated in lobar pneumonia.

"4. The cephalin cholesterol test is of little value in the African, but it is unlikely to be negative in cirrhosis.

"5. The $ZnSO_4$ turbidity is consistently and most markedly raised in cirrhosis. It is almost always raised, but to a somewhat lesser extent in primary carcinoma and liver abscess. It is not significantly elevated above the average African range in lobar pneumonia.

"6. The serum albumin is commonly reduced in all four conditions, but less markedly so in lobar pneumonia.

"7. Conversely, the total globulin is commonly raised in all four conditions, but less markedly so in lobar pneumonia.

"8. The alpha globulins are not raised in cirrhosis. They may be raised in primary carcinoma, and are very commonly elevated in liver abscess and lobar pneumonia.

"9. Changes in the gamma globulin are paralleled by the $ZnSO_4$ turbidity; a marked elevation occurring in cirrhosis, a somewhat less marked rise in liver abscess and primary carcinoma and usually little change being found in lobar pneumonia." *A. R. D. Adams*

SHAFEI, A. Z. **Puromycin Alone or in Combination with Phthalyl-Sulfathiazole in the Treatment of Intestinal Amebiasis.** *Antibiotic Med.* New York. 1959, May, v. 6, No. 5, 279–82. [12 refs.]

Puromycin (Stylomycin, Lederle Laboratories), 1 gm. daily for 14 days, was given to 23 patients with amoebic dysentery (6 acute and the remainder chronic relapsing cases); to 12 of these phthalyl-sulphathiazole (3 gm. daily) was also given.

4 of the 11 given the antibiotic alone relapsed (3 acute and 1 chronic case); only 2 of the 12 given the combined treatment did so. The author therefore concludes that the additional treatment materially improves the prospect of cure with puromycin treatment. *A. R. D. Adams*

SHAFEI, A. Z. **The Treatment of Amebic Dysentery with Paromomycin. A Preliminary Report.** *Antibiotic Med.* New York. 1959, May, v. 6, No. 5, 275–8.

Paromomycin (Humatin, Parke, Davis & Co.) is a broad-spectrum antibiotic which *in vitro* is said to equal or exceed the tetracyclines, erythromycin, chloramphenicol or neomycin in its antibacterial action. It cures experimental amoebic infections in rats and dogs; it is very well tolerated by them when given orally as absorption from the intestine is limited. When injected over a period of 3 weeks it may cause renal tubular degeneration in animals.

8 patients with acute amoebic dysentery and 12 during remissions of the disease were given 8·4 gm. of the drug orally in capsules over 7 or 14 days. All benefited clinically, with arrest of diarrhoea and of colon tenderness, and the stools of all were free from parasites within a week.

7 patients relapsed, with reappearance of parasites in the stools; only 2 of them were among the sufferers from the acute disease. Previous records of similar patients treated with other antibiotics are set out in a table for comparison. The author concludes that paromomycin at a dosage of 20 mgm./kgm. daily for 7 days cured 70% of cases of amoebiasis, and it caused no toxic side-effects. *A. R. D. Adams*

RELAPSING FEVER AND OTHER SPIROCHAETOSES

Del Vecchio, P. & Tresca, G. Rilievi clinico-terapeutici e considerazioni epidemiologiche e profilattiche sull'epidemia di febbre ricorrente del 1958 in Asmara. [**The 1958 Epidemic of Relapsing Fever in Asmara. Clinical and Epidemiological Observations**] *Igiene e San. Pubblica.* Rome. 1959, May–June, v. 15, No. 3, 213–20. [18 refs.] English summary (4 lines).

In 1958 an epidemic of relapsing fever started in Asmara (Eritrea) in January and lasted the whole year. 707 cases were diagnosed microscopically. The present report is divided into 2 parts, the senior author dealing with epidemiology, and Tresca with the clinical aspect.

Clinical. In general the symptoms were those generally found, though there was marked tenderness in the hypochondria; enlargement of the liver and spleen, when present, was not marked; haemorrhages were not often seen, and a rash was very rare. The most important feature was the marked effect on cerebellar function, a condition not hitherto reported, which seemed to occur mainly in those patients in whom the clinical condition was most severe and—less often—when large numbers of spirochaetes were found in the thick drop. In addition the plantar reflexes were never normal or symmetrical; the symptoms referable to the central nervous system disappeared after treatment. As modern antibiotics were lacking, treatment was by Neosalvarsan with, in 18 of the most serious cases, the addition of prednisone in doses of 30 mgm. the first day and 20 mgm. the second. This combined treatment was very satisfactory: fever disappeared on the second day and the patients' condition improved rapidly. Tresca thinks that prednisone had a definite protective action on the liver, which was not affected by the arsensical, notwithstanding the serious hepatic insufficiency in all the patients treated.

Epidemiology and prophylaxis. Relapsing fever is endemic in Eritrea and is louse-borne. In contrast to others, this epidemic was not really seasonal as it lasted more or less the whole year and reached its acme in August after which the incidence fell sharply in September, though 20 cases were still reported in December. The marked rise first in July and then the August peak was not only associated with the rains but was especially associated with the advent of thousands of persons from the neighbouring Ethiopian area of Tigrai where the disease is hyperendemic. These persons, after the harvest of prickly pears, brought them to the Eritrean markets for sale. The vendors were very lousy and in fact many were admitted to hospital suffering from relapsing fever. On the return home of the harvesters the incidence began to fall rapidly. The case mortality rate was 9%. Intensive anti-louse measures were instituted, including the disinfestation of 5,000 houses and 20,000 of the townspeople, while roads were blocked and over 33,000 visitors to the town were dealt with by means of an anti-louse powder containing 15% DDT and 2% Gammexane. *W. K. Dunscombe*

LEPROSY

In this section abstracts are arranged as far as possible in the following order:—epidemiology, aetiology, pathology, diagnosis, clinical findings, treatment, control.

FRAGA DE AZEVEDO, J., GÂNDARA, A. F. & FERREIRA, A. P. Estudo de alguns casos de lepra na Província de Timor. [**A Study of some Cases of Leprosy in the Province of Timor**] *Anais Inst. Med. Trop.* Lisbon. 1958, Mar., v. 15, No. 1, 219–23, 1 fig. English summary (6 lines).

The number of patients suffering from leprosy as registered in 1955 by the Health Service of the Portuguese Province of Timor (Indonesia), was 119 in a population of 223,250 distributed among 21 localities. The highest number (92) was in Baucau. In 3 localities 24 patients were examined and of these 6 were classified as lepromatous, 1 as tuberculoid, 1 as dimorphous, and the rest as indeterminate. The authors consider that the incidence is at least 0·8 to 1·9 per 1,000 of inhabitants. *Ernest Muir*

RUSSELL, D. A. **Notes on Leprosy in the Territory of Papua and New Guinea.** *Internat. J. Leprosy.* Wash. 1959, Jan.–Mar., v. 27, No. 1, 26–30.

The Territory of Papua and New Guinea has a population of 1,779,128 out of which the known cases of leprosy are 2,272 and the estimated number 6,000 to 8,000. Of the known patients 2,036 are resident in 11 leprosaria. The present policy is to adopt selective segregation. The type rate per 100,000 of the population is: lepromatous 27, tuberculoid 99, borderline 0·84, indeterminate 0·13. The chief obstacles to control are the backwardness of the people and the difficulties of communication. *Ernest Muir*

BRAND, P. W. **Temperature Variation and Leprosy Deformity.** *Internat. J. Leprosy.* Wash. 1959, Jan.–Mar., v. 27, No. 1, 1–7.

Over 2,000 reconstructive operations on limbs and face gave the author the opportunity to study the distribution of leprosy in various tissues of the body. He found a chronic inflammatory granulomatous infiltrate in a great variety of tissues: connective tissue, tendon sheath, tendon, joint capsule, synovial membrane, bone, cartilage, nerve and nerve sheath, skin, mucous membrane, testes and lympth nodules. Damage extended down from the surface from 5 mm. to 1 or 2 cm. according to the severity of the disease. The author came to the conclusion "that it is variation in temperature that determines the susceptibility of a tissue to damage from leprosy". This would account for the degree to which the nose, ear, testes (the only true gland affected) are attacked by leprosy.

A special study was made of the pattern of motor and sensory loss in the disease. In addition to a superficial and patchy anaesthesia secondary to the involvement of sensory nerve endings, there is a severer paralysis of large nerve trunks causing total anaesthesia and motor paralysis which always affects the same nerves in the same places. In the early stages paralysis is a block to conduction but without loss of continuity of nerve fibre, and this is caused by

ischaemia secondary to oedema and cellular infiltration. In a limb with widespread disease " every nerve within a certain critical distance of the surface of the skin is infiltrated, and . . . at equal depths from the skin thick nerve trunks suffer more damage than thin ones ".

There are 2 independent factors operating: the infiltration of the nerves causing the swelling around the fibres is conversely proportional to the depths from the skin ; and, given an equal degree of infiltration, the thinner the nerve trunk the less it is likely to be paralysed. Infiltrated nerves near the skin may escape paralysis because they are thin, and deeper thick nerves escape because they are not infiltrated.

The same dependence for attack on proximity to the surface is seen in tendons, bone and cartilage. Soft downy hairs with follicles near the surface are first attacked ; eyebrow hairs with deeper follicles are affected later, while the hair of the scalp with very deep follicles generally escapes.

It is suggested that " there is probably an optimal temperature for the growth and activity of the leprosy bacillus, and that this optimum is just below body temperature, perhaps only 2 or 3 degrees ".

This observation may be taken into account by those attempting to reproduce leprosy in experimental animals. *Ernest Muir*

LUNDIN, F. E., Jr. & ROSS, Hilary. **Liver Dysfunction in Leprosy. Results of the Bromsulfalein Retention Test in 150 Cases.** *Internat. J. Leprosy.* Wash. 1959, Jan.–Mar., v. 27, No. 1, 43–7.

It had been suggested that because the bromosulphthalein (BSP) test measures excretion by parenchymal cells it should be one of the more useful procedures for the evaluation of liver function in leprosy, in the absence of jaundice and renal impairment.

The test was applied in 150 patients, 140 with the lepromatous and 10 with the tuberculoid type of the disease. Of the 150 patients 21 (14%) had an abnormal degree of dye retention, 17 of these being lepromatous and 4 tuberculoid. However, the fact that 15 lepromatous cases without jaundice and with negative BSP showed a positive icteric index (13·3%) may indicate that liver damage is more common than can be measured by the BSP test. A patient with Lucio leprosy showed a dye retention of 7·5% on admission, but this gradually diminished to negative as the patient improved. It is considered from this and another case that retention of the dye during an acute lepromatous infiltration is suggestive of liver impairment by generalized disease.

Ernest Muir

GUINTO, R. S. & WADE, H. W. **Results of Tests with Serial Dilutions of Lepromin in Separate Groups of Normal Young Children with a Comparison of Two Lepromins and the Dharmendra Antigen.** *Internat. J. Leprosy.* Wash. 1958, Oct.–Dec., v. 26, No. 4, Pt. 1, 328–45, 1 fig. [29 refs.]

In order to ascertain the effect of dilution of lepromin, and whether high dilutions would be practicable for field testing, two series of tests were arranged, one in a town and the other in out-lying villages, the subjects being healthy schoolchildren of from 6 to 9 years of age. In the first series there were 5

groups of an average of 116 children each. In the second series (of village children) there were 5 groups averaging 92 in each. In the first series two antigens prepared in different ways (M and W) were used diluted 1 to 1 and the W antigen was also used in 1 in 10 and 1 in 20 dilutions; the antigens were compared with each other and with Dharmendra antigen. In the second series the W antigen was used in dilutions of up to 1 in 80.

The results are shown in a series of 7 tables and a graph. In the first series the early (Fernandez) reactions with the W lepromin were only half the number in the second series (13·5 against 26·1%). The M stock antigen gave fewer early reactions than the W, suggesting that boiling may be less effective than autoclaving in freeing the soluble antigenic elements. The Dharmendra antigen was ineffective in very young subjects. There was a drop from 80·9 to 21·4% of Mitsuda positives in the first series between the 1 in 1 and the 1 in 10 dilution, and in the second series a similar drop from 91·4 to 40·0. It is concluded that a dilution as high as 1 in 10 would be entirely unsuitable for routine testing in field work.

[This valuable paper is so full of data that it is difficult to abstract adequately, and interested readers are advised to study the original.] *Ernest Muir*

CHAUSSINAND, R. Le problème de la nature et de la signification de la réaction à la lépromine de Mitsuda. [**Nature and Significance of the Mitsuda Reaction**] *Ann. Inst. Pasteur.* 1959, Aug., v. 97, No. 2, 125–34. [18 refs.]

The author, in discussing the nature of the lepromin reaction, mentions that at the time when chaulmoogra was the only form of treatment for leprosy certain patients at first reacted positively to lepromin but later became insensitive to lepromin. This occurred without the patient's general condition being weakened, in fact sometimes the skin and nerve lesions went on improving. It was only after some months or years that the benign form of leprosy suddenly transformed itself into the malignant form.

The author and other workers have found that inoculation of lepromatous patients with BCG sometimes results in a feeble reaction to the lepromin test; but this only lasts for a short time and is not accompanied by any amelioration of the disease, and should therefore be regarded as without immunological significance.

However the author still considers that, because of the close affinity of the mycobacteria of tuberculosis and leprosy, it is possible that para-allergy to the leprosy bacillus in subjects infected with tuberculosis or vaccinated with BCG may be connected with a certain degree of resistance to leprosy infection. This matter can be ultimately cleared up only by clinical observation.

Ernest Muir

DOULL, J. A., GUINTO, R. S. & MABALAY, M. C. **The Origin of Natural Reactivity to Lepromin. The Association between the Mitsuda Reaction and Reactions to Graded Doses of Tuberculin.** *Internat. J. Leprosy.* Wash. 1959, Jan.–Mar., v. 27, No. 1, 31–42. [26 refs.]

The authors refer to their previously reported studies on Mactan Island where 23% of children from 6 to 35 months, 65% from 7 to 9 years, and 90%

of adults had given a positive late reaction to the lepromin test [this *Bulletin*, 1956, v. 53, 65, 324; 1958, v. 55, 410]. The questions arise as to what extent this increased reactivity with increased age is the result of infection with leprosy and tuberculosis, to what extent it is the result of previous injections of lepromin, and to what extent positive reactivity is the result of some other factor than infection with *Myco. tuberculosis* or any related species.

The authors give in tabular form the frequency of reactivity to lepromin in tuberculin-positive, compared with the frequency in tuberculin-negative persons, as shown in 11 different reports, in all of which the former was higher. The relationship was poorest where the dose of PPD had been least, but there was no obvious trend to larger differences as the dosage was increased.

A further study was considered necessary "in which the same lots of tuberculin and of lepromin would be used for all the subjects tested". Out of 868 children of 6 to 10 years of age 27·3% reacted to 10 TU of tuberculin, and of these 89·9% also reacted to lepromin, while of the nonreactors 72·3% reacted to lepromin, a difference of 17·6%. Although there is a positive correlation between reactivity to tuberculin and reactivity to lepromin, the excess of lepromin reactors among tuberculin positive subjects over the number expected if there were no association, comprises a very small fraction of the total number reacting to lepromin. This suggests some other factor than mycobacterial infection, and even to add the sensitizing effect of the test dose of lepromin would not be adequate without assuming an increasing capacity to respond to lepromin as age advances.

It is felt that further studies of lepromin reactivity are necessary. In order to confirm the opinion that patients with tuberculoid leprosy are almost universally lepromin positive, comparison should be made with healthy siblings, and the groups should be matched carefully in respect of age and sex. The theory should be tested that increasing reactivity with age is simply a "maturation" phenomenon. *Ernest Muir*

Viel, B. & Dellien, H. Relaciones entre lepra y tuberculosis. [**The Relationship between Leprosy and Tuberculosis**] *Acta Med. Costarricense*. 1958, Aug., v. 1, No. 3, 167–76. English summary.

The existence of leprosy in Argentina, Bolivia and Peru, countries bordering on Chile, and a constant migration from these countries to the Chilean mines and from other countries by sea, made it natural to expect the presence of leprosy in Chile. But search in the hospitals and laboratories, and studies by dermatologists who had studied leprosy elsewhere, have failed to discover the disease, with the exception of a few patients with tuberculoid leprosy who arrived in the country with the disease already developed. In Chile there are all kinds of climatic and social conditions with various densities of population. There is a great deal of poverty and tuberculosis is common.

To find out if the remarkable absence of leprosy is due to the presence of tuberculosis, tuberculin and lepromin reactions were tested in 499 primary schoolchildren in Santiago. The percentage of tuberculin-positives was 68·3, and lepromin-positives 72·8. Of these, 62·6% had both reactions positive, and 20·7% had them both negative; in 10·7% Mantoux was negative and Mitsuda positive, and in 6% Mantoux was positive and Mitsuda negative. The BCG vaccination of 68 children with both reactions negative resulted after 60

days in a conversion to positive of Mantoux in 79% and of Mitsuda in 75%. The authors conclude that the Mitsuda-positives are due to contact not with leprosy but with tuberculosis, that absence of leprosy in Chile may be due to the prevalence of tuberculosis, and that " where leprosy is still a problem, its eradication may be hastened with BCG vaccine ". *Ernest Muir*

YANAGISAWA, K. **The Effect of BCG Vaccination upon the Occurrence of Leprosy in Nursery Children.** *Internat. J. Leprosy.* Wash. 1958, Oct.–Dec., v. 26, No. 4, Pt. 1, 325–7.

Children of leprous parents, 248 in number, were divided into 3 groups: (1) those who were tuberculin positive on admission; (2) those tuberculin negative to begin with but who had been repeatedly inoculated with BCG at 1- or 2-year intervals; (3) those tuberculin negative to begin with but who had not received BCG inoculation. The leprosy morbidity rates in these 3 groups were, respectively, 5·1%, 1·5%, and 47·2%. With 1 exception the contacts of these children had been lepromatous leprosy patients.

The author draws attention to the fact that in tuberculosis, benefit from BCG can be expected only when the vaccination is received before the infection, while the above experiment shows that the children had probably been infected with leprosy before receiving BCG. Thus, because of the comparatively slow bacterial multiplication and immunity formation in leprosy, prevention of leprosy can be attained by BCG vaccination even when the vaccination is done after infection. *Ernest Muir*

RODRIGUEZ, J. N. **Relapses after Sulfone Therapy in Leprosy of the Lepromatous Type.** *Internat. J. Leprosy.* Wash. 1958, Oct.–Dec., v. 26, No. 4, Pt. 1, 305–12.

At the Culion Sanitarium in the Philippines there were over 5,000 patients before the war. There are still about 1,500 bacteriologically positive patients. The records show that the large majority of these receive irregular and insufficient treatment. " The main aim of the patients in receiving the treatment appears to be to minimize involvement of the eyes and the larynx and to avoid ulceration of the skin lesions, but not to become negative which would make them liable to be sent back to their homes." The relapse rate during a period of 3½ years was 7·0% under 6 months, 13·0% from 6 months to 2 years, 1·9% over 2 years, with an average relapse rate of 4·8%. At the Central Luzon Sanitarium with an average population of 2,100, where the treatment is better and the disease on admission less advanced, the relapses after similar periods were 3·0, 6·6 and 3·9%. Here again there was considerable absenteeism from treatment. Among 101 negative patients followed up by mobile clinics 3 had relapsed.

It is concluded that there is urgent need for accurate surveys of relapsed patients over a period of up to 10 years. Relapses would be greatly diminished if treatment were to be continued among discharged negative patients. All lepromatous type patients should have a minimal period of sustained treatment for 5 years whether bacteriologically negative or not. *Ernest Muir*

NAYLOR, R. F. **A Study of the Action of Sulfones on the Metabolism of Mycobacteria.** *Internat. J. Leprosy.* Wash. 1958, Oct.–Dec., v. 26, No. 4, Pt. 1, 313–17.

Using a Warburg respirometer, the author found alcohol to be slowly metabolized by *Myco. phlei*, and its oxidation markedly inhibited by DDS. But further respiration studies in no case showed specific inhibition by DDS to be demonstrable. Measurements of total growth were made by optical density methods; DDS inhibition was not very apparent, and it is concluded that the effect of DDS is largely bacteriostatic and not bactericidal. Experiments with *Myco. phlei* suggested that *p*-aminobenzoic acid reduced the inhibiting action of sulphone.

Following the experiments of HANKS [no reference given] showing that measurement of the dehydrogenase activity of *Myco. lepraemurium* provides a reliable index of the ability of the bacteria to infect animals, the author tested this method with *Myco. leprae*. But the results were negative, possibly because of the failure of the tetrazolium molecule to penetrate the lipid layer of the bacteria. However, dehydrogenase may diffuse from the bacteria and react with tetrazolium salts in the surrounding medium. The results obtained to date make it possible to detect by this method changes in the metabolic activity of *Myco. leprae* after DDS treatment. But further experiments are necessary to establish this finding on a quantitative basis. *Ernest Muir*

DAVEY, T. F. **Progress with New Antileprosy Drugs.** *Internat. J. Leprosy.* Wash. 1958, Oct.–Dec., v. 26, No. 4, Pt. 1, 299–304.

The author describes his method of using DDS as a control in testing new anti-leprosy drugs by "bringing together the records of as large a number of patients as possible who have had continuous DDS treatment, and producing graphs showing the average decline in bacterial index during DDS therapy at the center concerned". Beginning with a graph covering the progress of 150 patients over 4 years, the standard is improved year by year as more patients are included in it. There are also separate graphs for each degree of positivity up to a maximum bacterial index of 4·0 [the graphs are not reproduced in this paper].

It has also been found useful to record the condition of the bacilli, as successful chemotherapy is accompanied by an increasing proportion of degenerate forms of the lepra bacillus in routine smears. It has been noted that the reappearance of normal bacilli at sites where previously only degenerate forms could be found also gives warning of exacerbation of the disease which may result from drug resistance.

With this DDS standard control as above, 3 new anti-leprosy drugs have been tested recently: thiourea compound Ciba 1906 (DPT) [this *Bulletin*, 1958, v. 55, 524], diaminodiphenyl sulphoxides (DDSO) [*ibid.*, 1957, v. 54, 831], and diethyldithiolisophthalate (ETIP). DPT has been found useful in patients who have developed intolerance to DDS. It combines well with DDS and INH, but with doubtful additive effect. The only toxic effects were mild hypothyroidism in 2 patients on large and prolonged dosage. The large dosage and high price make it unsuitable for mass treatment.

DDSO is at least as effective, and at least as toxic as DDS.

ETIP [see *ibid.*, 1959, v. 56, 1146] is not suitable for administration by mouth or by injection. A twice-weekly inunction of 3 to 6 cc. has been found in some patients to exert a powerful effect on the bacilli, not only at the places of inunction but throughout the body. "Signs of degeneration are evident in bacilli within three weeks of the beginning of treatment, and a decline in bacterial index of as much as 1·3 is seen within three months in some individuals."

It is hoped to shorten treatment materially by using a succession of such drugs as these which have been tested against the DDS standard.

Ernest Muir

COOK, J. **Some Aspects of Reconstructive Surgery in Leprosy.** *East African Med. J.* 1959, July, v. 36, No. 7, 410–13.

LEITE, A. S., RÉ, L., SOBRAL, F. da C., PINTO, A. & SANTOS, A. Relatório da Missão de Prospecção à Lepra em Angola. [**Account of the Work of the Commission for Leprosy Inspection in Angola**] *Anais Inst. Med. Trop.* Lisbon. 1958, Mar., v. 15, No. 1, 335–56, 3 maps & 42 figs. on 21 pls.

The Report begins with an account of the formation of the Commission, the preparations made, and the route followed as shown in a map.

An account is given of the patients observed at 7 centres in the District of Huambo, which is the central and most highly populated district in Angola. The number was 901 out of a population of 37,009. The types of leprosy are given as: lepromatous 16·7%, tuberculoid 11·1%, indeterminate 72·2%. [From the photographs given it would appear that some of those classified as "indeterminate" are cases usually classified as tuberculoid.] In other districts which were visited the type rates were similar to the above.

As regards treatment, 3 different methods are recommended, depending on the nature of the country and its communications, and on the concentration of patients. (1) Where the population is dense and communications are open during the whole year exclusive ambulatory treatment should be adopted, injections being given at *postos sanitários* every 3 or 4 weeks. (2) When there are large numbers of patients far away from any *posto sanitário,* centres should be chosen where groups of patients can come together on fixed days and be treated by agents travelling by motor cycle. (3) Where the population is sparse and the communications are liable to be interrupted, patients should be grouped in small villages in sanitary conditions.

The leprosarium at Cazombo under a Protestant Mission is described. Such a place should be used for patients who need to be in hospital or cannot be rehabilitated. It should also be used as a centre for training workers and for research.

Ernest Muir

WARDEKAR, R. V. **Modern Approach to Leprosy Control.** *J. of J.J. Group Hosps. & Grant Med. College.* Bombay. 1959, July, v. 4, No. 3, 153–9.

The author describes the method of leprosy control by means of survey, education and treatment. Beginning with 1 unit in 1951 he increased the number of units to 10 by 1955, with a total population of about 200,000 under

observation. He found that the yearly surveys of these units helped early detection of new cases, that the early treatment prevented deformities, that out of 2,624 patients there was arrest of disease without residual signs in 37·9%, that in a follow-up of lepromatous cases under treatment for 1 to 6 years, from 21·1 to 77% became non-infectious in proportion to the length of treatment.

The plan of control recommends that all Government and Municipal medical officers, and those in charge of dispensaries, should be trained in leprosy work, and that leprosy patients should be treated along with other dispensary patients. In areas with high leprosy incidence where there is no general dispensary, control units should be established: " . . . there will be a central state organisation which will plan, guide and organise work but treatment will be completely decentralised. . . . Existing colonies will be used more and more for cases who need a short-term special medical care, they will be more like hospitals than like asylums ". [For a previous report of this work see this *Bulletin*, 1957, v. 54, 47.] *Ernest Muir*

BINFORD, C. H. **Histiocytic Granulomatous Mycobacterial Lesions produced in the Golden Hamster (*Cricetus auratus*) inoculated with Human Leprosy. Negative Results in Ten Experiments using other Animals.** *Internat. J. Leprosy*. Wash. 1958, Oct.–Dec., v. 26, No. 4, Pt. 1, 318–24.

In an attempt to inoculate leprosy bacilli and produce leprosy in experimental animals, some 1,500 small animals and 31 monkeys were used in 35 experiments. They were divided into 5 groups with, respectively: irradiation, irradiation plus cortisone, cortisone, inoculation only, and, as a control, heat-killed inoculum. The inocula consisted of nodules from lepromatous patients in the Philippines which were preserved by freezing. Only in golden hamsters were positive results obtained.

In another experiment 5 groups of golden hamsters divided as above were inoculated with leproma suspension in the testes and the ears. The two groups receiving cortisone died quickly. In the first group with irradiation only granulomas were demonstrated in 2 of the 10 animals, and in the fourth group with inoculation only granulomas were demonstrated in 6 animals. No lesions were found in the other groups. The first granuloma was shown 24 weeks after inoculation, but there was no demonstrable increase in bacilli till 18 months after inoculation, by which time practically all the histiocytes contained numerous bacilli. Transfer inoculation was made from the testis of one of these animals, but the results are not yet available.

A later experiment with golden hamsters similar to the above has shown the formation of granulomas in several of the animals.

In all these lesions the " uniform histiocytic composition of the granulomas in these animals, and the paucity of other inflammatory cells, affords a close parallel to developing lesions of human lepromatous leprosy ". Intraneural invasion by bacteria was noted in a few instances. Ultimate evaluation must await further study. *Ernest Muir*

HELMINTHIASIS

In this section abstracts are arranged as far as possible in the following order:—TREMATODES (*schistosomes, other flukes*); CESTODES (*Diphyllobothrium, Taenia, Echinococcus, other cestodes*); NEMATODES (*Hookworms, Ascaris, Filarial worms, Dracunculus, etc., Trichuris, Enterobius, Trichinella, etc.*).

LÝSEK, H. Příspěvek k úloze půdy a zeleniny v epidemiologii geohelmintóz. [**A Contribution to the Study of the Role of Soil and Vegetables in the Epidemiology of the Geohelminthiases**] *Českoslov. Epidemiol., Mikrobiol., Imunol.* Prague. 1959, v. 8, No. 2, 137–40. English summary.

In an investigation of the incidence of helminthiasis in the area of Jesenice, Czechoslovakia, examinations were made of samples of earth and of groups of 1–3 vegetables (carrots, parsley and cucumber) collected from the gardens of 9 families. The gardens had been manured once or twice each year with untreated human and animal excreta. The earth samples contained 10–3,160 helminth eggs per kgm., and up to 7 eggs were found in washings from the vegetables. *D. J. Bauer*

MAZIDIS, S. P. & DADIOTOU, D. D. [Sur un cas de bilharziose vésicale à *Schistosoma haematobium*] [**A Case of** *Schistosoma haematobium* **Infection**] *Acta Microbiologica Hellenica.* Athens. 1959, Jan.–Apr., v. 4, Nos. 1/2, 44–56, 2 figs. [In Greek.] French summary.

The following is a free version of the authors' summary:—

The authors report a case of *Schistosoma haematobium* infection in an Egyptian male aged 19, who had lived for 1½ years in Athens.

They comment on the danger that the disease may be established in Greece by Greeks returning home after residence in Egypt and the Levant, among whom carriers may be common; the snail host, *Bulinus contortus*, is known to exist in certain parts of Greece.

They suggest that particular attention should be paid to microscopic examination for eggs of the urine of people coming from the countries mentioned above, particularly in cases of haematuria. *Charles Wilcocks*

SANSARRICQ, H. La bilharziose à *Schistosoma haematobium* en Haute-Volta dans la région de Bobo-Dioulasso. [**Schistosomiasis due to** *Schistosoma haematobium* **in the Upper Volta in the Region of Bobo-Dioulasso**] *Méd. Trop.* Marseilles. 1959, May–June, v. 19, No. 3, 345–9.

The author studied the incidence of schistosomiasis in the south-west Upper Volta region, a plateau intersected by streams and pools and marshes and inhabited by agriculturists and fishermen, the children and adolescents often being in contact with the waters of the area. Records of the medical services showed only a low incidence of vesical schistosomiasis, except in the Houndé area.

The method used was to examine the sediment of centrifuged terminal urine passed after 5 minute exercise, 3 preparations being examined before a patient was declared negative.

From April 1956 to March 1957, 829 children from 26 villages were examined at 9 schools and a general incidence of 11·9% of positives was found, and 4 cases of microscopic haematuria without the presence of trematode eggs; but the most striking result was the exceptional frequency of eggs in the urines of pupils at the school at Houndé, where, out of 101 urines examined, 67 (66·3%) showed eggs and there were 3 cases of microscopic haematuria without eggs. Elsewhere the incidence was much less, varying from nil at Fò to 9% at Satri and Kotédougou.

The author refers to the findings of MARILL who reported 31·2% positives in 1,308 urines of children in the Houndé area [this *Bulletin,* 1957, v. 54, 1424].

Seeking further foci of schistosomiasis in the Bobo-Dioulasso and Orodara areas, the present author, having found very numerous snail intermediate hosts (*Bulinus, Pyrgophysa* and *Biomphalaria* alone or together) in a pool quite near the village of Santidougou, 20 km. north-east of Bobo-Dioulasso, examined urines of 75 inhabitants of this village after physical exercise and found *S. haematobium* eggs in 36 (48%). He then found an important focus of snails in the Canton of Séguédougou, north of Orodara, in a small, slow river with numerous villages near it, in which the people often fish. Here he examined, in May–June 1957, 832 people from 18 villages (about 5·7% of the total population of the Canton). Children chiefly were examined, *i.e.*, 475 boys and 314 girls, aged 5–15, and 31 adolescents or adult men and 12 women. In these 832 people he found eggs in 98 (11·7%), not counting 13 cases of microscopic haematuria without eggs. But in 8 villages no egg-carriers were found. In the others the incidences of infections varied from 2% to 66·6%. As Marill [*loc. cit.*] found in Houndé, the incidence was higher the nearer the villages were to the water from which the infection came.

The author therefore concludes that there is an important focus of schistosomiasis in the Houndé area and that, although an enquiry had shown that outside the Houndé area the incidence is low, or was, in one village, absent, further enquiry revealed 2 new foci of the disease, one at Santidougou, extending probably to villages on the neighbouring waterways, and another in the Canton of Séguédougou. *G. Lapage*

ALVES, W. **Further Studies on Antimony Dimercapto Succinate (TWSb) in Urinary Bilharziasis.** *Central African J. of Med.* 1959, June, v. 5, No. 6, 291.

The author refers to his previous account of the treatment of African schoolboys suffering from urinary schistosomiasis with TWSb [this *Bulletin,* 1958, v. 55, 665]. He has now treated 46 young Africans aged 17 to 21 years, at an industrial school near Salisbury, Southern Rhodesia, who were passing large numbers of viable *Schistosoma haematobium* and red blood cells in the urine. The patients were ambulant throughout.

To a bottle containing 2 gm. of the crystalline powder of TWSb 20 cc. of sterile saline was added. The dose was 5 cc. (0·5 gm. of TWSb) daily for 3 days. 20 patients received this total of 1·5 gm. of the drug intravenously and 26 received it intramuscularly. The solution was all used up on the day of preparation.

There were no serious side-effects: 3 patients in the intravenous group had

excessive salivation and 7 in the intramuscular group complained of pain, which was not serious, at the injection site.

Follow up was carried out at weekly intervals for 3 months: it was interrupted thereafter by the Easter vacation, but will be resumed. One patient in each group was positive throughout and a third in the intravenous group became positive at the sixth examination although he had been negative before that. There were thus 3 failures among the 46 patients treated.

The author concludes that the drug is fairly easily prepared, easily administered and appears to be without side-effects in the doses used, irrespective of whether it was given by the intravenous or intramuscular routes. He suggests that it would be desirable to attempt to reduce the course to 2 injections on successive days. If this were successful in achieving a cure it would " surely point the way to successful mass treatments of populations ".

He notes that FRIEDHEIM *et al.* [*ibid.*, 1955, v. 52, 56] were unable to detect untoward effects on the circulatory system of 134 patients treated with TWSb [for *S. mansoni* infections]. *H. J. O'D. Burke-Gaffney*

PELLEGRINO, J., BRENER, Z. & POMPEU MEMORIA, J. M. **A Comparative Study of Intradermal Tests and Stool Examination in Epidemiological Surveys on Schistosomiasis mansoni.** *Amer. J. Trop. Med. & Hyg.* 1959, May, v. 8, No. 3, 307–11, 2 figs. [15 refs.]

The authors, working in the State of Minas Gerais, Brazil, studied the relative efficiency of stool examinations and intradermal tests with cercarial and with adult worm antigens for diagnosing *Schistosoma mansoni* infections among (a) 454 unselected schoolchildren aged 10 to 15 years, and frequently exposed to infection, and (b) 558 unselected military personnel with only occasional exposures.

In the first group (children) a single stool examination showed 65·6% positive, whereas intradermal tests with cercarial antigen showed 57·6% positive, and with adult worm antigen 55·0% positive.

In the second group (adults) 24·19% showed *S. mansoni* eggs after a single stool examination, whereas the skin test was positive in 52·8% and 51·4%, with cercarial and adult worm antigens respectively. [It is of course clear, although not expressly stated in the paper, that whereas the finding of eggs is positive evidence, the results of the skin tests can only be regarded as presumptive evidence.]

The authors conclude: " The need for further studies in different epidemiological circumstances, using skin test and stool examination under standardized conditions in order to evaluate the real efficiency of these methods in epidemiological surveys, is emphasized ".

[This interesting and important paper should be read in conjunction with previous papers by the same authors, most of which have been abstracted in this *Bulletin*.] *R. M. Gordon*

PARAENSE, W. L. & DESLANDES, N. **The Renal Ridge as a Reliable Character for separating** *Taphius glabratus* **from** *Taphius tenagophilus*. *Amer. J. Trop. Med. & Hyg.* 1959, July, v. 8, No. 4, 456–72, 26 figs. [17 refs.]

" This paper is based on the comparative study of 15,516 specimens of *Taphius glabratus* from 36 localities and 12,776 of *Taphius tenagophilus* from 31

localities. A description of the renal region of both species is given, and it is concluded that they are distinguishable by the presence (in *T. glabratus*) or absence (in *T. tenagophilus*) of a ridge along the middle line of the membrane which covers ventrally the renal tube.

"The ridge makes its appearance in specimens of about 10 mm. in shell diameter and then gradually develops as the animal grows. It rises from a pigmented stripe which represents its forerunner in the small specimens under 10 mm. in diameter.

"The renal ridge is defined, both anatomically and histologically, and is shown to be a reliable diagnostic character of *T. glabratus* subject to age variation.

"The unridged renal membrane of *T. tenagophilus* is also defined, with especial reference to its variation and to some unusual characteristics that may lead the taxonomist to confuse *T. tenagophilus* and *T. glabratus*."

Authors' footnote:—

"Following the Rules of Nomenclature we use the generic name *Taphius* as the senior synonym of *Biomphalaria*, *Tropicorbis*, *Platytaphius*, *Afroplanorbis* and *Australorbis*. The species name *tenagophilus* refers to the large carinate Brazilian planorbid, until recently called *nigricans* by us; this nomenclature problem will be discussed elsewhere."

HAMILTON, P. K., HUTCHISON, H. S., JAMISON, P. W. & JONES, H. L., Jr. **The Pathology and Pathogenesis of the Hepatosplenic Disease associated with Schistosomiasis. ("Egyptian Splenomegaly")** *Amer. J. Clin. Path.* 1959, July, v. 32, No. 1, 18–33, 17 figs. [29 refs.]

Wedge biopsy specimens of the liver were taken from 73 Egyptian patients suffering from Egyptian splenomegaly while splenectomies were being done on them. In all but 10 of these patients the presence of a schistosomal infection [presumably with *Schistosoma mansoni*] was established microscopically. The histological changes found in the liver specimens were classified into 5 groups. Group I (26 cases) consisted of pipe-stem portal fibrosis; Group II (19 cases) coarse nodular cirrhosis; Group III (15 cases) diffuse portal cirrhosis; Group IV (10 cases) focal portal cirrhosis; and Group V (3 cases) included "miscellaneous"—2 patients had portal fibrosis with amyloidosis, and the third had subacute focal hepatic necrosis of schistosomal origin. Photographs and photomicrographs illustrate the features of these lesions, and there is amplification of the findings in the text. Dead adult worms were found in sections in 7 cases (some from each group), and schistosome ova were recognized in 74% of the liver biopsy specimens. "Schistosomal" pigment—dark brown pigment resembling haematin—was seen in 53% of the liver biopsy specimens; there was an evident relation between its presence and the finding of ova in the specimens.

In 49 cases in which macroscopic abnormalities were specifically sought for within the abdomen, gross abnormalities of the liver were evident in all; the gall bladder was abnormal in 71% of them, usually in the form of enlargement which often was considerable; there was evidence of portal-caval venous collateral circulation in 49%; schistosomal tubercles were seen in the omentum, mesentery, or colon serosa in 57%; and ascites was present in 69%.

In those cases where particular attention was paid to visible changes in the spleen perisplenitis, increased trabecular prominence, or Gamna-Gandy bodies (siderotic nodules) with haemorrhages were noted in more than half the patients; capsular thickening, or absence of Malpighian bodies in focal areas, were observed in less than half of them; and splenic infarcts were seen in 8 of the 49 patients. The weights of the spleens removed ranged from 550 to 2,420 gm.; all of them showed fibrocongestion, both macroscopically and microscopically. No schistosome ova were found in any of the spleens examined, and " schistosomal " pigment was found in them inconstantly. There was no consistent histological evidence of " hypersplenism ".

The authors conclude that schistosomal infection is the major aetiological factor in the liver disease occurring in Egyptian splenomegaly. Intrahepatic thrombopylephlebitis and peripylephlebitis (caused by dead worms and by live ova) is the origin of the liver disorder. Chemotherapy may precipitate these changes. Dietary deficiency probably renders the liver more liable to damage. The portal hypertension and splenomegaly are the result of obstruction of the intrahepatic veins and the formation of fibrous septa and anastomoses between the portal and hepatic veins. *A. R. D. Adams*

Polak, M., Montenegro, M. R., Meira, J. A., Conte, V. P., Espejo, H., Franchini, F. & Pontes, J. F. **Schistosomiasis mansoni. Pathological Changes in the Liver in Different Stages of the Disease studied by means of Laparoscopy and Needle Biopsy.** *Rev. Inst. Med. Trop. São Paulo.* 1959, May–June, v. 1, No. 1, 18–40, 16 text figs. & 8 coloured figs. on 2 pls.

Accounts of the pathology of schistosomiasis due to *S. mansoni* hitherto have been based on material obtained from advanced cases of the disease by liver biopsy or post-mortem examinations. The authors have endeavoured to study its progress through all its stages by photographic laparoscopy and needle biopsies in conjunction with the clinical, radiological (including splenoportography), oesophagoscopic, sigmoidoscopic and laboratory findings. The 30 Brazilian patients they report on were all suffering from *Schistosoma mansoni* infections but, as far as could be determined, from no other past or present disease of consequence (88 others were excluded on this account). None of them, with a single exception, had previously been treated for their schistosomiasis, to infection with which they had been exposed from infancy.

The intra-abdominal organs were examined directly by a " Foto-Laparoskop " of German manufacture and records were made by colour photography. Liver biopsy material was taken with a Vim-Silverman needle at the same time. One patient died of haemorrhage from a ruptured oesophageal varix 3 weeks after the examination, and after treatment with Repodral. All relevant data are set out in tables; there are good photomicrographs and there are excellent colour reproductions of the laparoscopic picture. On the appearances of the liver the progress of the disease could be divided into 4 stages. In Group I (9 patients) the liver if enlarged was only slightly so; its anterior edge was sharp; its surface was smooth and brilliant; and its colour red or reddish brown. In 5 of the patients whitish spots were seen on the liver surface; in 1 instance there were adhesions to the diaphragm. Biopsy revealed in all some slight periportal cellular infiltration and fibrosis, pigment deposition, and liver

cell degeneration and regeneration; in 6 cases granulomata were found, 3 of them contained *S. mansoni* eggs. Most of these 9 patients suffered from dysentery periodically, and there were visible changes in their rectal mucosae.

In Group II (9 patients) the liver was sometimes moderately enlarged; its edge was similar to that seen in Group I patients, and its colour brown or reddish brown but the surface was irregular or rough and furrowed. The liver capsule was opaque over these depressions. The histological appearances broadly were similar to those of the preceding group. Asthenia and dysentery were usual among the patients of this group.

In Group III (10 patients) the liver was enlarged and deformed; the edge was irregular and dented; the surface was grossly nodular with furrowed or flat depressions which often were whitish in colour, though the nodules were brown or reddish brown. Histologically there was usually marked lobular disarray with much periportal and intralobular fibrosis, Kupffer cell reaction, black pigment deposition, and evidence of liver cell degeneration and regeneration. In all patients in this group there was splenomegaly, often very great; the spleens were reddish or dark brown, sometimes with thick fibrous patches and adhesions to the great omentum. These patients all had oesophageal varices; enlarged veins could be seen within the abdomen though not externally. The clinical findings included oedema of the lower limbs (6 patients), ascites (7 patients) and haematemesis (4 patients).

In Group IV (2 patients) the liver was big, deformed and coated with fibrous tissue; it showed the histological changes already noted but in more marked form. The spleen was enlarged and sheathed in a fibrinous coating; oesophageal varices and intra-abdominal collateral circulation were visually evident, and there were ascites and oedema of the lower extremities.

The authors conclude that in the progress of *S. mansoni* infection the liver develops surface depressions and furrows which increase in extent and number until the surface becomes grossly nodular. The histopathological picture differs in various parts of the liver; laparoscopy gives a better assessment of the changes as a whole than does needle biopsy. Fibrosis of the portal fields is the outstanding and constant pathological change, and this may extend into the lobules. Kupffer cell proliferation is equally constant; the cells contain dust-like granules, and sometimes haemosiderin. The presence of schistosomal granulomata and ova may be revealed on biopsy; on autopsy of the only patient who died dead adult worms were found occluding vessels, and these were surrounded by a cellular exudate. The changes as a whole conformed to SYMMERS's clay-pipe-stem cirrhosis or HASHEM's coarse periportal fibrosis [this *Bulletin*, 1947, v. 44, 730]. Liver function tests showed some degree of liver impairment, especially in the last 2 groups of patients; no jaundice was observed in any of them. *A. R. D. Adams*

OKABE, K. & TANAKA, T. **A New Urine Precipitin Reaction for Schistosomiasis japonica. A Preliminary Report.** *Kurume Med. J.* 1958, v. 5, No. 2, 45–52.

In infections with *Schistosoma japonica* the diagnosis and the assessment of treatment may be difficult since eggs may be difficult to find. In acute infections a month elapses before eggs appear and in chronic cases the discharge of

eggs is irregular. Concentration methods are complicated and expensive. For several months after treatment some patients continue to discharge eggs.

Immunological methods such as the intradermal, precipitin, flocculation and complement-fixation tests have been investigated with antigen made from eggs, cercariae and adult worms. Okabe *et al.* [see this *Bulletin*, 1955, v. 52, 661] show that precipitin reaction with antigen made from adult worms was positive in acute infections with a titre of 1 in 6,400, and in chronic cases 1 in 800.

A precipitin reaction done with a patient's urine is positive and becomes negative after treatment. The condensed and dialysed urine of 22 patients with a chronic infection showed positive reactions with the serum from rabbits infected with schistosomes in titres of 1 in 4 and 1 in 16. The serum from the same patients gave positive precipitin reactions with titres ranging from 1 in 800 to 1 in 3,200 with antigen made from adult worms. There was no correlation between the serum and urine precipitin reactions. 5 patients were treated with TWBs and followed up by faecal examination and serum and urine precipitin reactions: the serum precipitin reaction remained positive while the urine reaction became negative as the eggs disappeared. *W. E. Kershaw*

SADUN, E. H., WALTON, B. C., BUCK, A. A. & LEE, B. K. **The Use of Purified Antigens in the Diagnosis of *Clonorchiasis sinensis* by means of Intradermal and Complement Fixation Tests.** *J. Parasitology.* 1959, Apr., v. 45, No. 2, 129–34, 1 fig. [10 refs.]

After referring to 4 earlier papers on this subject [see, for example, this *Bulletin,* 1957, v. 54, 317], the authors record their own results obtained with purified antigens made from adult *Clonorchis sinensis* collected from rabbits experimentally infected 2 months earlier with 800 to 1,000 metacercariae each, the latter having been obtained by pepsin digestion from fish collected in endemic areas in Japan. The adult trematodes, after repeated washing in physiological saline and distilled water, were put in ampoules, frozen and lyophilized. From these worms 4 antigens were prepared: a relatively crude fat-free antigen (CC), a lipid-free, borate buffer extraction (CTP), an acid-soluble protein fraction (CM) and an acid-insoluble but alkali-soluble protein fraction (CM-ins). CC was prepared by the method of CHAFFEE *et al.* [*ibid.*, 1955, v. 52, 173]. The other 3 antigens were prepared by the method of MELCHER [*ibid.*, 1944, v. 41, 225] for the preparation of *Trichinella spiralis* antigens. Protein determinations, standardization of antigens, performance of the complement-fixation and intradermal tests were done as described by Sadun *et al.* (*Amer. J. Trop. Med. & Hyg.*, 1959, v. 8, in press). The control solution for the intradermal tests was a buffered physiological saline containing 1 in 10,000 merthiolate [thiomersal] (BSM).

Preliminary tests showed that all positive intradermal tests were of the immediate type and reached their peak about 15 minutes after injection, when the limits of the weals were measured as described by Sadun *et al.* (*loc. cit.*) and the standards of positivity and negativity were those described by Sadun *et al.* in schistosomiasis (*Amer. J. Hyg.*, 1959, v. 68, in press). Intradermal tests were done on people from endemic areas in Korea and Japan who had proved clonorchiasis and also harboured various intestinal helminths and protozoa. The controls were people from non-endemic areas who were negative

by stool examination for the eggs of *C. sinensis*. For the study of cross-reactivity of the intradermal tests the authors used Koreans with paragonimiasis or Japanese with schistosomiasis (*S. japonicum*). Sera for the complement-fixation tests were obtained from patients with proved clonorchiasis and from trematode-free controls and to test the degree of specificity of this reaction the authors used sera from patients with tuberculosis, leptospirosis, virus hepatitis, paragonimiasis and schistosomiasis. Rabbit sera were used to study the development of antibodies in experimentally infected rabbits.

The intradermal tests showed that the CM antigen gave the most reliable and consistent results and this was used for tests on 395 persons. Of these all but 3 of 132 (98%) infected with *C. sinensis* gave positive intradermal tests, all but 2 of the 120 controls being negative. Cross-reactions occurred in 53 of 93 (57%) patients infected with *Paragonimus westermani,* and in 5 of 50 patients infected with *Schistosoma japonicum* ; but the weals produced by the homologous antigens were always larger and more distinct and could be distinguished from weals produced by homologous and heterologous antigens used concurrently. The authors think that the use of this purified antigen (CM) will eliminate most of the false positives and negatives reported by HUNTER *et al.* (*Military Med.,* 1958, v. 122, 85). Furthermore, clonorchiasis is usually distinguishable clinically from schistosomiasis and paragonimiasis.

Because most of the patients were adults with symptoms of clonorchiasis, other tests were done on 356 children aged 8 to 14, presumably without symptoms, from a school in an area in Korea where clonorchiasis was epidemic. Table II shows that in these, the results of the intradermal tests and of single stool examinations done by the formalin-ether technique were closely correlated, except that 38 children with eggs in the stools gave negative intradermal tests, while 10 without eggs in the stools gave positive intradermal tests. There were no significant differences in the results obtained with males and females, but, as fig. 1 shows, in both sexes the mean size of the weals seemed to increase with age and larger weals were obtained in patients with symptoms attributable to " liver fluke infection ".

For the complement-fixation tests the CM-ins and CTP antigens seemed to be the most sensitive. As table III shows, 23 out of 25 of the sera from patients with clonorchiasis gave a positive complement-fixation reaction and all but 1 of the 20 uninfected controls were negative. No false positives were obtained with patients with leprosy or virus hepatitis, but apparently false positives were obtained in 4 out of 21 patients with *Schistosoma japonicum,* in 19 out of 56 with *Paragonimus westermani,* in 10 out of 58 with tuberculosis and in 2 out of 8 with leptospirosis, but all the sera from patients with tuberculosis and leptospirosis were old. As the stools of these patients could not be examined, it was not possible to find out whether any of them had clonorchiasis or infection with *Metagonimus yokogawai* and storage of the sera may have affected the results.

To study the development of antibody response to *C. sinensis* 9 rabbits were infected and the results obtained with CTP and CM-ins antigens were compared. Table IV shows that 9 pre-infection rabbit sera reacted at low titres with CM-ins antigen, but not with CTP antigen ; otherwise there was no difference between these antigens. Maximal reactivity occurred 90 to 150 days after infection and thereafter antibody levels decreased. 2 rabbits artificially immunized with CC and CM-ins antigens gave titres of 1 in 16 and 1 in 32,

respectively, when tested with CM-ins antigen. Before inoculation the rabbit sera were negative.

[See also this *Bulletin*, 1959, v. 56, 852.]

[The references to the papers by CHAFFEE *et al.* and by MELCHER are incorrectly cited in the authors' list.] *G. Lapage*

TRIOZON, F. Diagnostic de la distomatose hépatique humaine par la recherche des oeufs dans les selles. [**Diagnosis of** *Fasciola hepatica* **Infection in Man by Examination of the Stools for Eggs**] *Ann. Inst. Pasteur de Lille.* 1957, v. 9, 105–12.

The author describes 22 cases of infection with *Fasciola hepatica* in the departments of Lyon, Saône-et-Loire and Rhône. The Janeckso and Urbanyi method of egg concentration (*Allatorvosi Lapok*, 1932, v. 17, 247) by flotation in a solution of mercury biniodide and potassium iodide was used and compared with other methods of concentration—with centrifugation and flotation in zinc sulphate solution and in salt solution. The mercuric iodide method recovered more eggs than the others and disclosed light infections which were not found by other methods. [The details can be found in the original.]

W. E. Kershaw

GIBSON, T. E. **The Identification of** *Cysticercus bovis,* **with special reference to Degenerate Cysticerci.** *Sanitarian.* London. 1959, Sept., v. 67, No. 12, 508–10, 2 figs.

"1. A survey of the cysts encountered in routine meat inspections, which macroscopically appeared to be *Cysticercus bovis*, revealed that only 25% were viable *C. bovis*. 4% were shown not to be *C. bovis*, and the remaining 71% were either caseous or calcified.

"2. Histological examinations of 18 caseous and calcified cysts revealed the presence of cestode tissue in only 3 of them. This proportion is too small to justify the use of histological methods as a routine measure.

"3. The difficulties for the meat inspector which these findings create are briefly discussed."

WOLFF, F., SCHNABEL, R. & MOLDENSCHARDT, H. Tödliche Atebrinintoxikation bei Bandwurmkur mittels Duodenaldusche. [**A Fatal Case of Mepacrine Poisoning during the Treatment of Tapeworm Infection by the Intraduodenal Route**] *Ztschr. f. ärztl. Fortbildung.* 1959, June 15, v. 53, No. 12, 683–7. [Numerous refs.]

The following is a translation of the authors' summary:—

This is the first observation of a death from overdosage of mepacrine [8·0 gm.] by the intraduodenal route in the treatment of tapeworm infection. The lethal operative dose absorbed amounted to 0·08 gm./kgm. body weight in a strong and healthy patient aged 19 years. The whole amount of the mepacrine was introduced within a period of about 1 hour.

Clinical and pathological findings, in agreement with animal experiments [HECHT, this *Bulletin*, 1934, v. 31, 171] and from study of the literature, indicate

a reversible toxic side-effect of mepracrine with a cerebral localization. There however is no doubt about its continued use as an anthelmintic in the usual therapeutic doses. *H. J. O'D. Burke-Gaffney*

GORDADZE, G. N. & GIGITASHVILLI, M. S. **[Epileptoid Fits provoked by** *Hymenolepis nana*] *Med. Parasit. & Parasitic Dis.* Moscow. 1959, v. 28, No. 4, 430–34. [In Russian.]

The English summary appended to the paper is as follows:—

"Epileptoid fits represent a grave manifestation in the clinics of hymenolepidosis. Judging by 26 cases observed by the authors these fits occur mainly among children in the age group of 7–15 (62·3%). The fits proceed with full loss of consciousness (77%), partial loss of consciousness (11·5%) and without loss of consciousness (11·5%). Aura is observed in 50% of the cases, and as distinct from the epilepsy of non-parasitic origin in the majority of cases consists in the intensification of some particular symptom of hymenolepidosis, nausea, headache, or pains in the abdomen.

"Discontinuance of fits after specific anthelminthic treatment testify to their parasitic origin. The intensity and frequency of the fits are mainly related to the intensity of the infestation.

"It appears that Hymenolepis nana may serve as an exciting agent of the cortex either by the way of reflexes or by the discharge of toxins."

SCHWABE, C. W.; SCHINAZI, L. A.; KILEJIAN, A. **Host-Parasite Relationships in Echinococcosis. I. Observations on the Permeability of the Hydatid Cyst Wall** [SCHWABE]. *Amer. J. Trop. Med. & Hyg.* 1959, Jan., v. 8. No. 1, 20–28, 5 figs. [36 refs.] **II. Age Resistance to Secondary Echinococcosis in the White Mouse** [SCHWABE, SCHINAZI & KILEJIAN]. *Ibid.*, 29–36, 16 figs.

These two papers are the first of a series on the biological aspects of the host-parasite relationship in *Echinococcus* sp. infections, and on the physiology of *Echinococcus granulosus*.

i. Small hydatid daughter cysts of *E. granulosus*, obtained from bovine lungs and livers, were weighed and then immersed in solutions of varied tonicity and composition. The author's conclusions—which are given in full below, concerning the permeability of the laminated and germinal layers of the hydatid cyst wall—are based for the most part on changes in weight observed to have occurred in the cysts after immersion in the various fluids for specified time periods.

"1. The germinal and laminated membranes of the hydatid-cyst wall are readily permeable to water.

"2. The permeability of the laminated membrane to nonelectrolytes increases in order of decreasing molecular weight. $CaCl_2$, KCl & NaCl permeate the laminated membrane at approximately the same rate.

"3. The selective permeability of the hydatid-cyst wall breaks down rapidly when cysts are suspended in hypotonic solutions.

"4. Potassium ion and cyanide ion, when present in hypertonic media, cause a pronounced withdrawal of the germinal membrane from the laminated membrane suggesting an effect upon permeability.

"5. Urea penetrates the laminated membrane readily but, since the germinal

membrane withdraws markedly in its presence, differential permeability is suggested.

" 6. Acetylcholine, physostigmine and iodoacetate are each antagonistic to potassium in that they prevent the withdrawal of the germinal membrane. It is suggested that a cholinesterase system may function in the osmotic regulation of the hydatid cyst.

" 7. Acetylcholine, physostigmine, iodoacetate, cyanide and fluoride do not affect the permeability of the laminated membrane.

" 8. Results obtained with hyaluronidase are equivocal. While it appears to increase the permeability of the laminated membrane to glucose, and possibly to mannitol, it has no effect upon urea or sucrose permeation."

ii. Two distinct, although interdependent, aspects of the development of the scolices of *Echinococcus granulosus*, when injected into the peritoneal cavities of white mice, are considered.

Scolices in process of vesicularization in young mice appear to undergo a development which may be regarded as similar to what occurs when they are cultured *in vitro* [and also apparently to what occurs in the early stages of development in the normal host]. Among the changes noted were an early increase in size due to the absorption of fluid, together with a migration of scolex cells to the periphery to form the germinal membrane.

The authors investigated the susceptibility of mice at various age periods to infection with *E. granulosus* after the injection of scolices into the peritoneal cavity. Their results are summarized as follows:

" 1. White mice 48 days of age or younger at time of inoculation were highly susceptible to an initial infection with scolices of *Echinococcus granulosus* when the latter were given intraperitoneally.

" 2. Mice 71 days of age or older were relatively resistant to an initial infection under similar conditions.

" 3. Four mice which were unweaned at time of inoculation died on the 10th and 11th days postinfection.

" 4. The few cysts which developed in older mice were often larger than any which developed in younger counterparts. The possibility of an ecological 'crowding effect' is suggested.

" 5. The difference in susceptibility to infection appeared to be related to the magnitude of the host's cellular response." *R. M. Gordon*

FARHAN, I., SCHWABE, C. W. & ZOBEL, C. R. **Host-Parasite Relationships in Echinococcosis. III. Relation of Environmental Oxygen Tension to the Metabolism of Hydatid Scolices.** *Amer. J. Trop. Med. & Hyg.* 1959, July, v. 8, No. 4, 473–8, 5 figs. [10 refs.]

GARCÍA DE ONÍS, F. Sobre un caso de quiste hidatídico de bazo. [**Hydatid Cyst of the Spleen. Report of a Case**] *Cirugía.* Madrid. 1959, Apr.–June, v. 5, No. 20, 96–8, 2 figs.

ANTINE, W. & ROTHENBERG, S. P. **Hydatid Cyst of the Liver rupturing into the Bile Ducts: a Case Report describing a Characteristic Triad of Symptoms.** *Ann. Intern. Med.* 1959, May, v. 50, No. 5, 1277–82, 1 fig.

The patient, who complained of urticaria, coryza and epigastric pain, was a 42-year-old male who had emigrated to the United States from Italy 7 years

previously. "Because of the history of repeated attacks of colic and jaundice, laboratory evidence of biliary obstruction and a palpable mass in the liver, laparotomy was deemed advisable. The preoperative diagnosis was common duct stone or hepatic metastatic neoplasm." At the operation it became apparent that the real cause of the trouble was a hydatid cyst. Precautions were taken to prevent the spread of infection, following the possible escape of cystic fluid into the peritoneal cavity, and to guard against an allergic reaction. The patient made an uninterrupted recovery. Retrospectively, the authors recognized the triad of biliary colic, urticaria and jaundice as that described by DEW in his classic monograph on hydatid disease [this *Bulletin*, 1929, v. 26, 691].

R. M. Gordon

YAMASHITA, M. [**An Epidemiological Study of Hook Worms in Southern Kyushu**] *Igaku Kenkyu*. Fukuoka. 1959, June, v. 29, No. 6, 1953–68, 1 map. [56 refs.] [In Japanese.]

The English summary appended to the paper is as follows:—

"For four years from 1955 to 1959 collective feces examinations of hookworms and treatment of the infected were carried out repeatedly in 18 villages in Southern Kyushu. The discharged worms were classified in two kinds, *Ancylostoma duodenale* (AD) and *Necator americanus* (NA).

"1. The infection rate of AD was 30%, and the average number of worms discharged was 6·5 per head; and the respective figures for NA were 46% and 31%, indicating intensive infestation of the latter.

"2. The infection rates by village ranged from 59% to 14% for AD and from 69% to 27% for NA. In general, AD predominates in mountain villages and NA in low flat places.

"3. In a small island (Chikushima) AD predominates in spite of predominance of NA on the opposite coast districts. In the island, arable land being scanty, most people are engaged in fishing. This fact suggests that the modus of infection of AD is mainly per os and that of NA is percutaneous.

"4. By experience of mass treatment of the infected it was noticed that for the purpose of reducing the infection rate, the treatment must be repeated at least every other year, an interval of four of five years being almost ineffective.

"5. The studies seem to justify the assumption that AD gets infection mainly per os and NA percutaneous; this conclusion agrees with the infection experiment of both kinds of hookworms on human bodies."

SATO, Y. [*Ascaris* **Infection of Infants**] *Igaku Kenkyu* Fukuoka. 1959, June, v. 29, No. 6, 1686–92. [16 refs.] [In Japanese.] English summary.

This is a note to the effect that 15·9% of 164 children (presumably in Japan) up to the age of 24 months were found to be infected with *Ascaris*, some of them were infected by the 7th month. Infection probably arises from contaminated house dust. Artificially-fed infants showed rather more infection than breast-fed infants.

Charles Wilcocks

SAZ, H. J. & VIDRINE, A., Jr. **The Mechanism of Formation of Succinate and Propionate by** *Ascaris lumbricoides* **Muscle.** *J. Biol. Chem.* 1959, Aug., v. 234, No. 8, 2001–2005. [43 refs.]

SINGHA, H. S. K. **Mesenteric Adenitis in Association with Ascariasis.** *Brit. Med. J.* 1959, Aug. 22, 220–23.

In this paper the author draws attention to certain surgical aspects of intestinal ascariasis in tropical climates where very heavy infections are common among the poorer elements of the population.

A surgical complication frequently met in the author's experience in Colombo is acute mesenteric adenitis, presenting as clinical features generalized abdominal colic, nausea or vomiting and pain, maximal in an ill-defined area on the right side of the abdomen, with tenderness usually in the right iliac fossa. The temperature is normal or slightly raised and on palpation of the abdomen guarding is usually found but no rigidity and a noticeable degree of distension of the lower abdomen is constantly seen.

Operative findings were remarkably constant in the author's series of 5 cases occurring within a period of about 1 year. There was a sticky yellow peritoneal fluid covering the intestine (but not the parietal peritoneum); numerous roundworms in the lower ileum; segmental spasm of the intestine and alternating portions showing moderate dilatation and enlargement of the mesenteric nodes. Operative intervention was limited to removal of the appendix in every case as a precaution, and a biopsy of the omentum and of a lymph node. A few days after the operation piperazine citrate was given as an anthelmintic, usually with marked improvement after the evacuation of the worms with the stools.

It is difficult, in the author's experience, to differentiate between acute mesenteric adenitis and appendicitis; but in any case operative treatment with the removal of the appendix is considered advisable followed by specific anthelmintic therapy.

Histological examination in all 5 cases of the series showed either a normal appendix or lymphoid hyperplasia of the organ. The lymph nodes, on the other hand, always showed reactive hyperplasia. The differential count in all 5 cases, taken after the operation, was normal, and the leucocyte count varied from 12,000 to about 15,000 per cmm. In an addendum the author refers to 2 more cases, diagnosed before operation. *E. S. Phipson*

MIJARES, M. S., LAMBERTI, J. A. & BRUNI CELLI, B. Granuloma mesenterico producido por huevos de *Ascaris lumbricoides*. [**Mesenteric Granuloma caused by Eggs of** *Ascaris lumbricoides*] *Rev. Latinoamer. Anatom. Patol.* Caracas. 1958, July–Dec., v. 2, No. 2, 133–37, 4 figs. on 2 pls.

The English summary appended to the paper is as follows:—

"A case is presented of a granulomatous lesion caused by aberrant localization of eggs of Ascaris lumbricoides in the mesentery Attention is called to this lesion because of the frequency of ascariasis in our country and because the lesion described is likely to be misdiagnosed if the pathologist is not aware of its existence."

[See also this *Bulletin*, 1959, v. 56, 551.]

MATSUMURA, T. & YUGAMI, S. **Critical Examination of the So-Called** *Ascaris* **Toxin from Viewpoint of Allergy. Preliminary Report.** *Gunma J. Med. Sci.* Maebashi. 1959, Mar., v. 8, No. 1, 61–3.

The pathogenic effects of infection with *Ascaris lumbricoides* have been ascribed to a toxin though they are inconsistent and many resemble allergic

reactions. Experiments were undertaken in infected and non-infected guinea-pigs to differentiate the possible toxic effects from those which could be produced by allergy.

Since ascariasis is common in Japan, in man and in pigs, experimental animals may readily become sensitized by latent infections, and animals were bred in air-conditioned rooms which had been fumigated with methyl bromide to kill *Ascaris* ova. The food was heated to 100°C. Animals were not used until they had been bred for several generations. Some were then infected with *Ascaris*.

In the infected animals the serum showed precipitins, tanned cell agglutinins and passive cutaneous anaphylaxis against *Ascaris* body fluid, injection of the fluid intradermally produced a local allergic reaction, while intravenous injection caused death from anaphylactic shock; the fluid caused the isolated intestine to contract, and oral administration caused contraction of the intact stomach and duodenum with congestion and haemorrhage, and administration by all these routes a rise in neutrophils and eosinophils in the peripheral blood, while no reactions were produced in uninfected animals. Embryonated eggs given by mouth to non-infected guineapigs produced no change in the blood picture, but produced a neutrophilia and eosinophilia in those previously infected, with a remarkable eosinophilic infiltration in the lung.

The phenomena hitherto believed to be due to a so-called *Ascaris* toxin are now considered to be allergic. *W. E. Kershaw*

PIKE, Eileen H. **The Effect of Piperazine Citrate on Hatching and Migrating Larvae of** *Ascaris lumbricoides*. [Research Notes.] *J. Parasitology*. 1959, Feb., v. 45, No. 1, 46.

The effect of piperazine citrate was tested on the newly-hatched larvae of *Ascaris lumbricoides* in the intestine of white mice and on the migrating larvae in the tissues of the intestine, liver and lungs, 40 female white mice 6 to 8 weeks old having been infected orally with 5,000 infective eggs cultured for 6 weeks in 1% formalin. The eggs were obtained from worms obtained from man. To test the effects of piperazine on the newly-hatching larvae, 10 mice were given 100 mgm./kgm. body weight of piperazine citrate by mouth one hour before infection with the eggs and thereafter the same dose hourly until 6 doses had been given. To test the effects on the migrating larvae 10 mice were each given a single daily dose of 300 mgm./kgm. body weight of piperazine citrate intraperitoneally, the first dose being given 24 hours after infection with the eggs, the doses continuing for 7 days. The remaining 20 mice were used as controls and were given an equivalent volume of distilled water in the same way as the drug was given to the others. All the mice were killed 10 days after infection with the eggs.

Larvae seen in brain squashes of all the mice were actively motile. Those found in pepsin digests of individual organs or of carcasses were nearly all dead, this being presumably due to the digestion process, because in squash preparations before digestion active larvae were seen. The means of the number and extent of the pulmonary haemorrhagic lesions determined by the BROWN and CHAN classification (*Am. J. Vet. Res.*, 1955, v. 16, 613) for the hatching-larvae group were: test 2, controls 4. The means of the number of

larvae recovered from each organ for test mice and controls respectively were as follows:

	spleen	lungs	liver	brain	kidneys	muscle	total
Test	1	8	18	0	0	6	33
Control	7	73	63	1	5	346	495

In both the test and control mice the same number (4 to 12) of hatched larvae was found in each direct saline smear of faeces collected during the fourth and eighth hour after infection with the eggs. For the migrating larvae group (2 controls of which died with complete lung consolidation) the range of the Brown and Chan classification was: test, 2 to 4, controls 2 to 4; and the means of the number of larvae recovered from each organ for test and control mice respectively were as follows:

	spleen	lungs	liver	brain	kidneys	muscle	total
Test	5	37	47	0	0·4	110	199
Control	6	32	69	0	0	104	211

Brown and Chan (*loc. cit.*) stated that piperazine hydrochloride given orally to mice before and after feeding them with infective eggs entirely prevented the hatching larvae from invading the blood streams and lungs; but in the present study piperazine citrate did not prevent the migration of all the larvae, though it reduced the number recoverable from the various tissues to only 7% of those recoverable from untreated controls. When, however, the piperazine citrate was given 24 hours after infection with the eggs, *i.e.* when the larvae had already reached the tissues, piperazine citrate had no appreciable effect.

G. Lapage

ISHIZAKI, T., BANDO, T. & KOBAYASHI, Y. **The Studies on the Fluid Environment of *Ascaris*; Influence of pH and Effect of Antibiotics and Antimycotics (Dehydroacetic Acid) in Fluid Media upon the Survival Length and the Duration of Normal Locomotion.** *Japanese J. Med. Sci. & Biol.* 1958, Aug., v. 11, No. 4, 223–33, 4 figs. [12 refs.]

The survival of *Ascaris suis in vitro* was not prolonged by the addition of any carbohydrates, proteins, amino acids, vitamins and pieces of liver to physiological saline, either unchanged or renewed frequently.

The accumulation of metabolites of *Ascaris* and the growth of bacteria changed the *p*H of the medium and produced turbidity. Dihydrostreptomycin and penicillin and dihydroacetic acid suppressed the development of microorganisms, and with a *p*H of between 6 and 10 a combination of the three substances increased the survival.

W. E. Kershaw

BEAVER, P. C. **Visceral and Cutaneous Larva Migrans.** *Pub. Health Rep.* Wash. 1959, Apr., v. 74, No. 4, 328–32. [23 refs.]

The organisms implicated include the larvae of certain flies and helminths. Among the latter the larvae of various species of ankylostomes have long been recognized as a common cause of "cutaneous larva migrans"; while more recently, the larva of *Toxocara canis* (a common round-worm of the dog) has been found responsible for many cases of "visceral larva migrans" in children

[this *Bulletin*, 1956, v. 53 (1365) and 1954, v. 51, 420]. In the latter instance a high proportion of the larvae enter the central nervous system and a considerable number have been found in the eye. "The tragic consequence of invasion of the eye is the development of a lesion which by its resemblance to retinoblastoma prompts the unnecessary removal of that vital organ."

[This paper contains nothing new but it is an excellent guide to, and summary of, the previous literature, in so far as the helminthological aspects of the subject are concerned.] *R. M. Gordon*

MANSON-BAHR, P. **The Story of *Filaria bancrofti*. A Critical Review. Part I. Early History.** *J. Trop. Med. & Hyg.* 1959, Mar., v. 62, No. 3, 53–61, 5 figs. [34 refs.] **Part II. Metamorphosis of *W. bancrofti* in the Mosquito and Filarial Periodicity.** *Ibid.*, Apr., No. 4, 85–94, 2 figs. [50 refs.] **Part III. Advances in the 20th Century.** *Ibid.*, May, No. 5, 106–17. [71 refs.] **Part IV. Mosquito Intermediaries of *W. bancrofti*.** *Ibid.*, June, No. 6, 138–45, 1 folding map. [53 refs.] **Part V. Description of *W. bancrofti* and Pathology of Filariasis.** *Ibid.*, July, No. 7, 160–73, 12 figs. [Numerous refs.]

This critical review is a much needed and long awaited account of the history of a helminth infection of such importance that many believe it to rank next to malaria among the parasitic diseases affecting man.

The compilation of this series of papers must have involved its author in many weeks of research in the literature, research of a type which could successfully be undertaken only by one who is constantly in touch with the progress of events and who has himself added much to our knowledge of the subject. The result is a solid piece of work which will serve as a reliable source of reference for many years to come. This scientific solidity, however, does not mean that the work is merely an unattractive menu of dates and facts; on the contrary, it is a delightful meal of five courses, flavoured with humour and served with the wine of comment for which its author is renowned.

To attempt further to abstract a work of reference which covers some 260 papers, varying in date from 1863 to the present day, would serve no useful purpose; one can only urge all who are interested in filariasis to read it in its entirety. *R. M. Gordon*

TOUMANOFF, C. Filariose humaine et sa transmission dans la Basse-Guinée (Estuaire du Rio Nunez). [**Transmission of Human Filariasis in the Rio Nunez Estuary Region of Lower Guinea**] *Bull. Soc. Path. Exot.* 1958, Nov.–Dec., v. 51, No. 6, 908–12.

An examination of the blood films of 250 inhabitants from 2 villages on the coast of French Guinea showed 12 infections (4·8%) with *Wuchereria bancrofti* and 5 infections with *Dipetalonema perstans*. Both *Anopheles gambiae melas* and *Mansonia uniformis* were captured in huts. As most of the mosquitoes captured were gorged, precipitin tests were possible and showed that most of the blood meals were from man. Out of 66 females of *M. uniformis* dissected, 3 had proboscis infections of filariae and 2 had filariae in the thorax. In *A. gambiae melas*, 1 female out of 35 dissected had filariae in the proboscis.

M. uniformis is noted as attacking man indoors and out-of-doors, mainly at night but also during the day. Females with partially digested blood meals have been found resting in huts. The breeding places of *Mansonia* were found only once, but the adult mosquitoes are widely distributed and are believed to be important vectors of human filariasis in this region. [In view of the presence of *Wuchereria* infections in animals in East Africa, transmitted by *Mansonia,* the presence of animal filariae cannot be ruled out, but see also this *Bulletin,* 1958, v. 55, 1260.] *B. R. Laurence*

JORDAN, P. **The Possible Rôle of Low-Density Microfilaraemia in the Spread of *Wuchereria bancrofti* by *Culex fatigans* in East Africa.** *Ann. Trop. Med. & Parasit.* 1959, Apr., v. 53, No. 1, 42–6. [10 refs.]

The following results were obtained from dissections of *Culex p. fatigans,* dissected for pre-infective and infective forms of *Wuchereria bancrofti* after feeding about 12 days previously on patients in Tanganyika who were variously pre-treated, or untreated, with diethylcarbamazine. An untreated person with low microfilaraemias (a mean of several microfilarial counts of 0·5; range 0 to 1 microfilaria per 20 cu. mm.) infects the mosquito (2·6% of 38 mosquitoes). Persons treated 4 hours and 1 day previously with 250 mgm. of the drug, with 500 mgm. 8 hours, 2 or 14 days previously, or with 1,000 or 1,500 mgm. (over 2 or 3 days) 2 or 3 days previously, were still infective to the mosquito, the blood films showing mean counts, over an hour at the time of feeding, of 0·5 to 13·1 microfilariae per 20 cu. mm. (about one-third of the counts were actually nil). Mosquitoes still developed essentially normal levels of infections of late developmental forms if infected first from an untreated person and then fed, a week later, on persons treated with the drug at 500 mgm. for 3 days, or at 5 and also one hour before the mosquitoes fed.

The results are compared with previously published data [this *Bulletin,* 1953, v. 50, 542; 1955, v. 52, 1112; 1956, v. 53, 466]. It is concluded that the present work emphasizes that mosquito infection follows from lower microfilaraemia levels than previously supposed; mosquito infection is normal from microfilariae persisting after treatment with diethylcarbamazine; advanced developmental stages in the mosquito are not affected by intake of the drug in a subsequent meal by the mosquito; and, finally, in eradication programmes persons with few microfilariae in the blood should be included in treatments with diethylcarbamazine. There is some preference for including also those having negative blood at examination. There is general recognition of the significance in this connexion of mosquito susceptibility to infection and biting rates. *D. S. Bertram*

OMORI, N. **Experimental Studies on the Role of the House Mosquito, *Culex pipiens pallens* in the Transmission of Bancroftian Filariasis. 3. Duration of Life of Filariae in Mosquitoes exposed to Winter Temperatures.** *Yokohama Med. Bull.* 1958, Dec., v. 9, No. 6, 382–90, 7 figs. [11 refs.]

Experiments to test the possibility of the survival of larvae of *Wuchereria bancrofti* in hibernating *Culex pipiens pallens,* kept at laboratory winter temperatures in the south of Japan (below 16°C.), have shown that the filariae,

at all stages in the mosquito, do not survive winter temperatures. Filarial larvae killed by low temperatures, especially when in stage I or infective, were often found encapsulated by black "chitin", but actively growing stage II larvae, which appeared to be the least resistant to low temperatures, tended to disintegrate under these conditions. Transmission of *W. bancrofti* filariasis in Japan through the hibernating mosquito does not seem to be possible.

B. R. Laurence

LINARD, R. L'éléphantiasis de la verge. Sa cure opératoire. [**Operative Treatment of Elephantiasis of the Penis**] *Ann. Soc. Belge de Méd. Trop.* 1959, Feb. 28, v. 39, No. 1, 95–100, 4 figs. (3 on pl.)

Compared with the common disease of scrotal elephantiasis in which the penis is involved, elephantiasis of the penis alone is unusual. It does occur, however, and when the penis becomes grossly enlarged there is severe impairment of function. After removal of the elephantoid covering, there remains the interesting problem of how to provide a new cover for the organ. Classically, a skin graft obtained with a Padgett dermatome is the method of choice, but this requires both instruments and technical skill that are not often available in the interior of the Congo. If the bare penis is left to cicatrize this is a tedious and slow process and leaves a poor result.

The following method has been found simple and effective. It consists of covering the penis with a fat-free pedicle skin graft from the thigh, and is completed in three stages. The skin just below the groin is thin and free from hair and there is a choice of side in case sepsis or large lymph nodes are present unilaterally. Stage 1 consists of pulling the bare penis through a tunnel in the upper part of the selected thigh. This tunnel has been made by two incisions, one at the level of the corona penis and one near its base as the penis lies obliquely across the skin selected. The incisions are at right angles to the long axis of the penis and the tunnel is made by undercutting between them. Haemostasis is secured by gauze pressure for 5 minutes. A few sutures fix the superficial surface of the penis to the skin where it enters and leaves the tunnel. A rubber catheter is inserted and the area bandaged firmly.

Stage 2 follows after 15 days or 3 weeks. Two extensions of the original incisions are carried down the thigh in the same line as before and far enough to construct a flap that will wrap around the rest of the penis. The flap is completed by an incision joining the ends of these two incisions and this flap is then ready to complete the covering for the penis. The distal half of the penis is freed completely from its tunnel by an incision along its upper margin, leaving the skin flap attached by only a pedicle over the proximal half of the penis. Tulle gras dressing covers the raw thigh area and the catheter is changed. After another 15 days, the penis is finally detached from the thigh, and the raw area there covered with Thiersch grafts if necessary. The catheter is now finally removed.

[An even simpler method, which has been uniformly successful, is to use Thiersch grafts of postage stamp size directly on to the bare surface of the penis and cover with tulle gras for 10 days. This leaves the penis free and no catheter is required. Movement of the penis does not hinder the "take" of these grafts which form an excellent covering for this organ. The grafts may be cut with a razor or scalpel.]

J. R. Rose

SAMS, W. M. & BECK, J. W. **Subcutaneous Filarial Infections. A Report of Two Additional Cases from Florida.** *Arch. Dermat.* 1959, Mar., v. 79, No. 3, 294–7, 3 figs. Discussion 297–8.

Two subcutaneous human filarial infections are reported from Florida. From an inflammatory cystic lesion in the cheek of a housewife a parasite resembling an immature female *Dirofilaria immitis* was recovered; and from a woman of 60 years old with a lump in the breast, granulomatous tissue was removed which, on section, contained a nematode which could not be identified. This may have been an infection with *D. immitis* or of one of the species of *Dipetalonema* normally infecting animals but occurring in this instance in man.
[See also this *Bulletin*, 1952, v. 49, 980.] *W. E. Kershaw*

BERTRAM, D. S. **Experimental Epidemiology with the Filarioid Infection of Cotton Rats.** Reprinted from *Proc. Tenth Internat. Congress of Entom.* 1956 (1958), v. 3, 503–6.

"Several forms of filariasis affect man in the tropics, some severely. The vectors are mosquitoes or other blood-sucking diptera, depending on the species of worm. Filarioid infections are also known from a wide range of animals and birds, but little is known about their vectors. Important exceptions are mosquito-borne filariases of dogs and mite-borne filariasis of cotton rats. Cotton rat filariasis has made practicable attempts at quantitative investigations of the inter-relationships of host, parasite, and vector. Initial work dealt with cotton rats infected once only with different numbers of worms, the ensuing infections, their infectivity for the vector, and the efficiency of the mite as a vector. Investigations were then developed on the more complex results of re-infection, artificial epizootics being arranged under laboratory conditions. Prolonged exposure to re-infection resulted in cotton rats of subnormal weight, associated with considerable infections of adult worms. But few or no microfilariae occurred in the blood-stream, thus disrupting, or jeopardising, the continuity of transmission. With intermittent transmission, the cotton rats were of normal weights, considerable adult worm infections were again evident, and the microfilarial infection of the blood was extremely high and persisted so for months. Transmission is assured, although less intensively than might be supposed. These two patterns are contrasting cases of an infinitely variable quantitative relationship between the host, parasite, and vector. They suggest that marked seasonal transmission, as opposed to all-year-round transmission, could be important to the apparent incidence and the epidemiology of, at least, cotton rat filariasis if not other infections in other hosts."

CROSSKEY, R. W. & CROSSKEY, M. E. **A Quantitative Survey of Onchocerciasis in Persons under Twenty Years of Age in an Endemic Area of Northern Nigeria. With a Consideration of the Epidemiology based on a Mathematical Hypothesis, by F. N. Macnamara.** *Ann. Trop. Med. & Parasit.* 1959, Apr., v. 53, No. 1, 10–24, 1 map & 6 figs. [13 refs.]

Wishing to obtain information on the relationship between the frequency and intensity of infection with *Onchocerca volvulus* and the distance of the patients' homes from the nearest breeding ground of *Simulium damnosum*, the authors have studied and recorded findings on 2,043 people between 3 and 20 years of

age, living in the Abuja Emirate of Northern Nigeria. A snip of skin was taken from the iliac crest of each and examined for microfilariae, the numbers being recorded on a simple system, while at the same time the home of the person and its distance from *Simulium* breeding places were noted. This material is presented for successive two-year age-groups for boys and girls living at 0–2, 2–6 and 6–10 miles from *Simulium* sources.

The figures are analysed by Macnamara, by means of an ingenious and acceptable technique, who shows that the risk of infection remains about the same throughout the age period studied, though it varies according to the place of residence. Only one introduction of infection is required to produce a low-grade intensity of infection in the iliac crest area, 4–7 introductions are necessary to produce the next higher grade recorded and perhaps 8–15 for the third grade utilized. The tabulated information shows that the frequency of these degrees of severity is related to the distance from potential sources of infection and it is found on analysis that the inoculation rate varies inversely with the distance [in a degree which would produce extinction of the risk at about 13 miles]. *G. Macdonald*

ISRAEL, M. S. **The Nodule in Onchocerciasis.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1959, Mar., v. 53, No. 2, 142–7, 8 figs. on 4 pls. [16 refs.]

70 excised onchocercal nodules were examined by histological and histochemical means and every nodule was found to contain a large proportion of disintegrating worms, whether or not the patients had previously been given treatment by Antrypol. Half of the nodules contained only dead worms: on this evidence it was suggested that healthy worms lie free in the subcutaneous tissues and that nodules are formed in response to products of dying worms, and that the nodule is not an essential part of the disease. [This belief, which may be true of old nodules, is not supported by the ease with which live microfilariae can be recovered by aspiration from a nodule. It seems likely that active adult worms may lie free in the tissues and later become encapsulated in the nodules, which, as has been suggested by DIAZ, may be regarded for most of their existence as the nest of the adult worms, only later to become their mausoleum.] *W. E. Kershaw*

PARAVISSINI, F., OLIVER-GONZALEZ, J. & MALDONADO, J. F. **Treatment of Massive *Trichuris trichiura* Infections with Telmid.** *Bol. Asoc. Med. de Puerto Rico.* 1959, Apr., v. 51, No. 4, 135–7.

"The results of this study indicate very strongly that Telmid [Dithiazanine iodide] is very effective for the treatment of infections with *Trichuris trichiura*. This is in agreement with the findings of other investigators [this *Bulletin*, 1958, v. 55, 1262]. In 30 out of 32 children the drug removed all of the worms as indicated by negative stools. In the remaining 2 the stools showed light egg counts.

"The drug caused no serious side effects except nausea and vomiting as observed in the hospitalized children. These reactions subsided once the treatment was ceased.

"These studies also indicate that therapy can be given at home with no inconveniences."

SOKOLINSKY, I. R. [**Combined Treatment of Trichocephaliasis in Children with Oxygen and Diathermy**] *Med. Parasit. & Parasitic Dis.* Moscow. 1959, v. 28, No. 4, 438–9. [In Russian.]

The English summary appended to the paper is as follows:—

"Oxygenotherapy in combination with diathermy as applied for the treatment of trichocephaliasis in children proves to be the most effective and accessible method of this kind. Of importance is the proper preparation of the patient, this, in a great measure, predetermining the success of the cure.

"Intrarectal introduction of oxygen is being stretched over 3 days. In the evenings, on the eve of each successive day, the patient is given a saline laxative, and 10–15 minutes prior to the oxygen administration a high purgative enema is instituted.

"At the time of the oxygen introduction the region of the large intestine is being massaged by stroking it counterclockwise. 15 daily sittings of diathermic treatment of the right iliac region of 30 minutes duration each are prescribed following oxygenotherapy, with laxative administered after the 8th and 15th sittings.

"Richardson's balloon is recommended for the dosage of oxygen, being suggested for this purpose by O. S. Mishchenko, but its design actually in use is modified by the author himself.

"Of 22 children subjected to this kind of treatment full recovery was noted in 21."

LABZOFFSKY, N. A., KUITUNEN, E., MORRISSEY, L. P. & HAMVAS, J. J. **Studies on the Antigenic Structure of** *Trichinella spiralis* **Larvae.** *Canadian J. Microbiol.* Ottawa. 1959, Aug., v. 5, No. 4, 395–403, 2 figs.

"By means of physical and chemical procedures seven antigenic fractions have been isolated from *Trichinella spiralis* larvae, as determined by complement fixation. Some evidence is presented to indicate that at least some of the isolated fractions are antigenically distinct and that antibodies in *Trichinella*-infested rabbit develop at different times for different fractions. Importance of using a properly selected antigen in serodiagnosis of trichinosis is discussed."

ZIMMERMANN, W. J., HUBBARD, E. D. & MATHEWS, J. **Studies on Fecal Transmission of** *Trichinella spiralis.* *J. Parasitology.* 1959, Aug., v. 45, No. 4, 441–5, 1 fig.

"Investigations on the fecal transmission of *T. spiralis* have been carried out using fox cubs and albino rats as donors, while swine, albino rats and fox cubs served as recipient hosts. The feces of experimentally infected animals have been examined by the Baermann technique to ascertain the number of larvae eliminated.

"The following factors were found to affect transmission: (1) time interval between infection and passage of feces by donor, (2) relative immunity of donor, and (3) number of trichinae ingested by the donor. About 95% of the larvae passing through the intestinal tract of the donors were recovered from

feces eliminated within 24 hours after ingestion. Immune animals eliminated a larger percentage of the administered trichinae than did nonimmune animals."

SUESSENGUTH, H. & BAUER, A. H. **Clinical and Serological Evaluation of 9,236 Suessenguth-Kline Slide Flocculation Tests for Trichinosis.** *Pub. Health Lab.* 1959, Mar., v. 17, No. 2, 33–40, 1 chart.

The sources of the 9,236 blood specimens examined and the results obtained with the authors' antigen [this *Bulletin*, 1958, v. 55, 200] may be summarized as follows:—

Source	Number of individuals examined	% Negative	% Weakly positive	% Positive
Cord bloods from infants	1,030	100	0	0
Persons suffering from some inflammatory disease	463	94·8	2·2	3·0
Persons infected with parasites other than *Trichinella*	105	92·4	3·8	3·8
Unselected persons whose blood had been submitted for routine serological testing	7,042	90·3	3·6	6·1
Persons in whom trichinosis was suspected	519	64·2	5·2	30·6
Persons in trichinosis epidemics	77	15·6	2·6	81·8

"Because of the time-lag between the infection and the appearance of the first positive reaction, repeated tests for at least 3½ weeks after infection are necessary before a negative reaction is unconditionally accepted."

R. M. Gordon

DEFICIENCY DISEASES

LUYKEN, R. & LUYKEN-KONING, F. W. M. **Nutrition Research on the Windward Islands (Netherlands Antilles). II. Medical Examination of the Population.** *Trop. & Geograph. Med.* Amsterdam. 1959, June, v. 11, No. 2, 103–14. [21 refs.]

This paper records the clinical examination of some 1,200 children. No clear-cut deficiency disease was found, but xerosis and folliculosis of the skin were common and slight liver enlargement frequent. Dental caries was widespread and associated with a high intake of sticky sweets and of white flour. A large part of the paper is taken up with records of height, weights and other physical measurements of the children. Skeletal age lagged behind real age by 6 to 12 months.

R. Passmore

RAOULT, A. Aspects de la malnutrition chez le grand enfant en A.O.F. Données de la ponction-biopsie hépatique (Suite I). [**Aspects of Malnutrition in the Child in French West Africa. I. Results of Liver Puncture Biopsies**] *Bull. Soc. Path. Exot.* 1959, Jan.–Feb., v. 52, No. 1, 97–114. (Données biochimiques.) Protéinémie et électrophorèse sérique (Suite II). [**II. Biochemical Findings. Blood Proteins and Serum Electrophoresis**] *Ibid.*, 114–25. [41 refs.]

i. This paper reports on the biopsies from 180 children aged 2 to 12 years. In 21 the liver was not enlarged, but in the remainder there was hepatomegaly and in 40 this was recorded as + + + or more. The histological picture was complicated. Fatty degeneration was found in only 6 cases and probably disappears rapidly after the age of 3. Marked hyperplasia of the Kupffer cells was common and also infiltration with lymphocytes and histiocytes. This was often associated with fibrosis, which was more severe in the older children. Vascular lesions, such as the occlusion of the central hepatic vein as described in Jamaica, were not seen. Malarial pigment was seen in almost all the livers of the younger children, but was less common in those 12 years of age. Thus the presence of pigment was not closely associated with fibrosis and malaria was not considered as an important cause of the chronic liver damage. Chronic malnutrition was closely associated with the fibrosis. Lack of dietary protein appeared to be particularly important. Enlargement of the parotid glands, almost certainly of nutritional origin, was frequently present. Schistosomiasis and infective hepatitis probably contributed to the fibrosis in some children; it was very difficult to get precise information about previous attacks of hepatitis. Malnutrition appeared to favour the development of fibrosis in a child after an attack of hepatitis, but it does not follow that the virus infection is an essential factor in cirrhosis in Africa.

[This excellent paper based on a very thorough study emphasizes the complexity of the factors responsible for cirrhosis of the liver in Africa. The main conclusion is that malnutrition is relatively more important than infections.]

ii. Figures for serum albumin and serum globulin are given for healthy children, children with enlarged liver, enlarged spleen, enlarged parotid glands and with the "syndrome polycarentiel". All of these conditions were associated with a marked increased value of gamma globulin and smaller increases of the other globulin fractions. Serum albumins were also low.

In a few children the levels of blood ascorbic acid were low, below 10 mgm. per litre. Serum calcium levels were also sometimes low and in 6 out of 111 children the level was 70 mgm. per litre. There was no clear association between the level of blood ascorbic acid and gingivitis or other signs of scurvy.

R. Passmore

DUVERNAY, C. E. & DELANOE, G. Contribution à l'étude des asystolies carentielles et organico-carentielles par ponction biopsie du foie. [**Use of Liver Biopsy in the Study of Asystoles due to Nutritional Deficiency alone or with Organic Heart Failure**] *Maroc Méd.* 1959, Mar., v. 38, No. 406, 432–55, 19 figs. [39 refs.]

See also this *Bulletin*, 1959, v. 56, 474.

LEITNER, Z. A. **Purpura in Kwashiorkor.** [Correspondence.] *Brit. Med. J.* 1959, Aug. 29, 303.

This correspondent refers to the paper by KAHN and STEIN [this *Bulletin,* 1959, v. 56, 1178] and criticizes the use of the word " purpura " in the absence of associated haematological changes. He points out that the presence of spots resembling purpura in connexion with protein malnutrition has been established in different parts of the world. He considers that " purpura-like spots " fully covers the description given by the authors. It does not imply non-existent haematological changes and " helps to avoid confusion about a syndrome which is now well defined and may be encountered and diagnosed all over the world ".

H. J. O'D. Burke-Gaffney

CHAKRABORTY, M. L. & CHATTERJEE, A. C. **Neutral 17-Ketosteroids in Children in Conditions of Malnutrition.** *Calcutta Med. J.* 1959, July, v. 56, No. 7, 223–7. [16 refs.]

" 1. The present study has found out the normal 17-ketosteroid output in the children of this part of the country.

" 2. Total excretion of 17-ketosteroids in children of ages from 1 to 8 is 0·84 mg. per 24 hours on average.

" 3. The present study has produced evidence that malnutrition is associated with hypofunction of suprarenal glands.

" 4. The present investigation presents logical explanation to the previous finding of one of the authors (M.L.C. 1944) that in oedema of malnutrition the chloride level of blood was low. Suprarenal glands thus play a significant role in producing oedema in malnutrition.

" 5. It also provides a cogent exposition of the report of M.L.C. 1948 & Gopalan *et al* 1952, that in conditions of malnutrition there occurs structural damage of the suprarenals."

FOOD AND AGRICULTURE ORGANIZATION OF THE UNITED NATIONS. **FAO Nutrition Meetings Reports Series No. 22. Report of the FAO/UNICEF Regional School Feeding Seminar for Asia and the Far East. Tokyo, Japan, 10–19 November 1958.** 52 pp., 1 diagram. Rome: 1959. [Sales agent for U.K., H.M. Stationery Office.] [$0.50 ; 2s. 6d.]

This report emphasizes the value of school meals in improving the health of children in the tropics. The administrative and financial aspects of school feeding are fully discussed. An appendix gives a useful account of local foods suitable for children, *e.g.,* saridele and tempeh (Indonesia), groundnut extract curd, Indian multipurpose food, miso and natto (Japan), fish flour and food yeast (Taiwan).

R. Passmore

HAEMATOLOGY

BHATNAGAR, D. P., GOVIL, K. K. & PRASAD, B. G. **Haematological Studies in Healthy Adults.** *Indian J. Pub. Health.* 1959, Apr., v. 3, No. 2, 169–76. [22 refs.]

"1. Total plasma protein values have been estimated in a group of 36 male healthy adults drawn from different areas of U.P. State by micro-Kjeldahl method. The values ranged from 7·17 gm. % – 8·31 gm. %.

"2. The subjects were clinically examined with record of age, height and weight; a dietary survey of the group was also made, the findings were recorded in schedules suggested by Indian Council of Medical Research for such surveys. The individuals were enjoying good health except for minor defects in few cases. The dietary intake was adequate in all respects except in vitamin A and C which is a normal observation in the case of rural dietetic survey.

"3. Different plasma protein moieties have also been estimated simultaneously. Albumin values range from 4·2 gm. % to 5·74 gm. %, Fibrinogen from 0·2 gm. % to 0·53 gm. %, globulin from 2·05 gm. % to 3·55 gm. % and A. G. ratio from 1·36 to 2·71, in the group.

"4. Haematological studies made are Haemoglobin, Total R.B.C., Total W.B.C., P.C.V., Differential W.B.C. and calculated values of M.C.V., M.C.H. and M.C.H.C.

"5. The significance of total plasma protein values and its different fractions in assessing the nutritional status of a person have been discussed."

SENEVIRATNE, R. D. de A., GUNASEKARA, D. B. & MAHADEVA, K. **An Experimental Investigation of Anaemia.** *Ceylon J. Med. Sci.* (Sect. D.) 1958, Feb., v. 9, Pt. 2, 69–79, 2 figs. [28 refs.]

"This work was undertaken to establish normal haematological standards for rabbits in Ceylon and to study how they react to haemorrhage and haemolysis.

"These standards formed the first step in the investigation of the role of sex in anaemias in rabbits.

"Normals for Ceylon rabbits have been recorded and compared to the normals of rabbits in other parts of the world, and the significance of the difference discussed.

"Effects of haemorrhage and haemolysis have been described and their significance in relation to anaemias discussed.

"The possible role of sex is briefly indicated."

CRADOCK-WATSON, J. E., FENTON, J. C. B. & LEHMANN, H. **TRIS Buffer for the Demonstration of Haemoglobin A_2 by Paper Electrophoresis.** *J. Clin. Path.* 1959, July, v. 12, No. 4, 372–3, 2 figs.

CHOREMIS, C., ZANNOS-MARIOLEA, L., AGER, J. A. M. & LEHMANN, H. **Persistence of Haemoglobin "Barts" beyond Infancy in a Child with Thalassaemia.** *Brit. Med. J.* 1959, Sept. 5, 348–9, 5 figs.

"In a 9-year-old child with thalassaemia haemoglobins A and F and also haemoglobin 'Barts' were found. Hitherto 'Barts' has been demonstrated

only in early infancy, and its persistence into a later age in conjunction with thalassaemia is further evidence in favour of its being a variant of foetal haemoglobin."

LATHEM, W. & JENSEN, W. N. **Plasma Hemoglobin-Binding Capacity in Sickle Cell Disease.** *Blood.* 1959, Sept., v. 14, No. 9, 1047–56, 3 figs. [21 refs.]

"Studies of the capacity of plasma proteins to bind hemoglobin were made in patients with sickle cell anemia (SS), sickle cell trait (SA), hemoglobin C disease and in patients with hemolytic anemias. Hemoglobin binding was quantitatively normal in sickle cell trait, but was greatly reduced or absent in sickle cell anemia, hemoglobin C disease and in other hemolytic disorders. These alterations have been attributed to a reduction in the level of hemoglobin-binding proteins in circulating plasma. The mechanism of this reduction was not established, but the observed changes were correlated with the presence of increased hemolytic activity.

"The binding of hemoglobin C and hemoglobin S by normal plasma was quantitatively normal."

VENOMS AND ANTIVENENES

KOCHWA, S., IZARD, Y., BOQUET, P. & GITTER, S. Sur la préparation d'un immunsérum équin anti-venimeux au moyen des fractions neurotoxiques isolées du venin de *Vipera Xanthina palestinae.* **[Preparation of an Anti-Venom Horse Serum by means of Neurotoxic Fractions isolated from** *Vipera Xanthina palestinae* **Venom]** *Ann. Inst. Pasteur.* 1959, Sept., v. 97, No. 3, 370–76, 1 fig.

"A potent antineurotoxin-containing serum against the venom of *Vipera Xanthina palestinae* was prepared in horses by immunization with electrophoretically separated neurotoxic venom fractions, subsequent to immunization with whole venom."

ADROUNY, G. A., DERBES, V. J. & JUNG, R. C. **Isolation of a Hemolytic Component of Fire Ant Venom.** *Science.* 1959, Aug. 21, v. 130, 449.

"A crystalline hemolytic principle, shown to be a constituent of fire ant venom and having the properties of an amine, was isolated from crude extracts of whole ants. The chromatographic procedure of isolation is described, and a preliminary report is given about some properties of the substance."

[See this *Bulletin*, 1957, v. 54, 1002.]

TOXOPLASMOSIS

CUBONI, E. L'intradermoreazione con toxoplasmina mei conigli immunizzati verso il *Toxoplasma gondii*. [**Intradermal Reaction to Toxoplasmin in Rabbits immunized against** *Toxoplasma gondii*] *Nuovi Ann. d'Igiene e Microbiol.* 1959, Mar.–Apr., v. 10, No. 2, 159–61.

Out of 24 rabbits repeatedly injected with killed toxoplasms, obtained from the peritoneal exudate of infected guineapigs, immunization was produced only in 8 animals, whereas none of the others survived. Intradermal inoculation of living parasites produced specific skin reactions in 6 of the immunized animals.

C. A. Hoare

FERRARA, A., ORLANDELLA, V., SANTORO, E. & ANDRINI, F. Tipologia ed interpretazione delle lesioni viscerali nella toxoplasmosi sperimentale. [**Types of Visceral Lesions in Experimental Toxoplasmosis**] *Giorn. di Malattie Infettive e Parassit.* 1959, Mar., v. 11, No. 3, 165–89, 30 figs. [46 refs.]

A critical account is given of the histopathology of toxoplasmosis in the light of the authors' investigations on experimental infections of guineapigs, mice and rabbits, supported by literary data. Descriptions are given of the pathological changes in the central nervous system, lungs, heart, liver, spleen and lymph glands, skin, alimentary canal and pancreas, kidneys and genital organs, with detailed protocols of the findings in individual animals, and numerous photomicrographs illustrating the lesions.

According to the authors' observations, toxoplasmosis affects a great variety of organs, with a special predilection for vascularized tissues. In this connexion it is noted that the preponderance of cerebral and ocular lesions is not due to particular tropisms of the parasite but is determined by its preference for the vascular elements of these organs. In the various tissues affected, the parasites invade the endothelial cells, especially in organs containing sinuses where the flow of blood is sluggish, whereas organs with rapid blood circulation (*e.g.* heart, large blood vessels) are usually free of parasites.

The pathological changes in toxoplasmosis are said to be of 3 types: (a) in the initial phase of the infection the lesions show cytolytic and necrobiotic changes, which are attributed either to an endotoxin produced by the parasite or to its products of metabolism; (b) pulmonary, hepatic and splenic lesions are characterized by proliferative and metaplastic changes of the type found in histiocytic elements; and (c) cases in which reparative and regenerative processes, varying according to the organs affected, are predominant.

The course of the disease also varies in different hosts: thus in rodents it is usually acute and fatal, with parasites invading diverse tissues; in large mammals (including man) there is every transition from symptomless to acute infection; finally, in birds (*e.g.* fowl) the infection may be generalized without producing visceral lesions.

C. A. Hoare

DERMATOLOGY AND FUNGUS DISEASES

AGRAWAL, S., SHARMA, K. D. & SHRIVASTAVA, J. B. **Generalized Rhinosporidiosis with Visceral Involvement. Report of a Case.** *Arch. Dermat.* 1959, July, v. 80, No. 1, 22–6, 5 figs.

The subject was a Hindu male, aged 30, who complained of a growth on the right lower eyelid and similar nodules all over the body. The epitrochlear and inguinal glands were enlarged and there was oedema of the feet. Biopsy specimens from a growth on the palate, and on the right leg, showed histological features diagnostic of rhinosporidiosis.

The patient died suddenly a few hours after a liver biopsy had been performed.

Nodules from skin, eye, palate, tongue, epiglottis and larnyx taken at autopsy all showed similar features. The subepithelial lesions were crowded with large sporangia, measuring 200μ–300μ in diameter. The organism was seen in various stages of its life cycle and many of the sporangia were ruptured, liberating spores into the tissues. Individual spores were 7–10μ in diameter. The lungs showed formation of granulomatous nodules, situated in the interalveolar septae and there was marked giant-cell reaction around sporangia and spores. These sporangia and spores were also found in liver and spleen where the cellular response was negligible and a few degenerate spores were present in the kidneys.

A somewhat similar case, which was reported by RAJAM *et al.* (*Indian J. Surgery*, 1955, v. 17, 269), would point to a haematogenous dissemination. In this instance spores of *Rhinosporidium seeberi* were demonstrable in the urinary sediment, peripheral blood and ascitic fluid.

A comparison is made between this infection and that of coccidioidomycosis, but the sporangia of *R. seeberi* are larger, and filled with spores smaller than those of *Coccidioides*, with uniformly-sized granules 1·5μ in diameter.

Philip Manson-Bahr

VANBREUSEGHEM, R. & VANDEPUTTE, M. Mycétome de la nuque chez un Noir du Congo belge. [**Mycetoma of the Neck in an African in the Belgian Congo**] *Ann. Soc. Belge de Méd. Trop.* 1959, Apr. 30, v. 39, No. 2, 227–38, 11 figs. on 4 pls. [13 refs.]

The English summary appended to the paper is as follows:—

"A mycetoma developed within 2 years on the back of the neck of an African boy 10 years old, living in Leopoldville (Belgian Congo). It was a white grain mycetoma due to a true fungus and is therefore a maduromycotic mycetoma. A fungus was isolated but was not identified because it produced only sterile hyphae. When inoculated into a rat's testis it caused a giant-cell reaction similar to that seen in the patient, but had not formed grains even after 8 months. Histologically there was a rather rare picture of grains in a giant plasmodium initially about 30μ in size subsequently reaching a final diameter of 400μ. The grains alone, without the plasmodium were very rarely seen. The authors discuss the significance of this phenomenon."

ALFONSO ARMENTEROS, J. & ROMERO JORDÁN, O. Una nueva forma clínica de la cromoblastomicosis. Forma seudo-queloidea. [**A New Clinical Form (Pseudo-keloid) of Chromoblastomycosis**] *Bol. Soc. Cubana de Dermat. y Sifil.* 1958, Sept.–Dec., v. 15, Nos. 3/4, 152–8, 5 figs.

The authors report a case in Cuba. The patient was an adult Negress and the disease started 4 years previously as a focus of intense itching on the dorsum of the left foot, without any preceding trauma. Within a few weeks there was a febrile reaction with lymphangitis, which recurred repeatedly at intervals of a month or more. The first lesions appeared on the foot and gradually the leg became covered with a large number of nodules, normal or red in colour, the largest being 3–4 centimetres in diameter. They were round or oval, raised, hard and fibromatous. Some seemed to contain fluid, but when opened produced only scanty serous or sanguinolent exudate. The lesions were usually (but not always) discrete; there were about 50.

Histologically there was marked hyperkeratosis with multiple micro-abscesses, containing greenish-yellow bodies in groups of 2 or 4, which showed a double contour; some of these were dividing. The dermis showed a tuberculoid condition with giant cells enveloping the parasites; there were oedema and fibrosis.

Cultivation on "Sabouraud glucosado" produced colonies in 22 days, grey-black in colour. Growth was much more slow than with other pathogenic strains of *Hormodendrum* with which the fungus was compared. Microscopic examination showed that growth did not resemble the *Hormodendrum* or the *Phialophora* types and the authors suggest the name *Acrotheca pedrosoi*.

The patient was given vitamin D2 and anti-anaemic treatment, but she disappeared. *Charles Wilcocks*

TROPICAL OPHTHALMOLOGY

CORNAND, G. Le trachome au Tidikelt occidental. [**Trachoma in the Western Tidikelt Region**] *Arch. Inst. Pasteur d'Algérie.* 1959, June, v. 37, No. 2, 315–28, 5 figs. on 2 pls. & 1 chart.

[See this *Bulletin*, 1959, v. 56, 495.]

DEREANI, C., JESE, L., JUNG, M., KMET, J. & MURRAY, E. S. **A Note on the Transmission of the Inclusion producing Agent of Trachoma by means of Material Stored at −60°C.** *Rev. Internat. du Trachome.* 1959, v. 36, No. 1, 106–12.

The authors produce evidence that the agent of trachoma present in conjunctival scrapings will retain its activity for at least 3 months when kept at −60°C. *S. P. Bedson*

TSUTSUI, J. **Achromycin and Achromycin-Hydrocortisone Oil Suspension in the Treatment of Trachoma.** *Rev. Internat. du Trachome.* 1959, v. 36, No. 1, 73–85.

The author has treated a large number of patients suffering from trachoma with tetracycline [Achromycin] alone and tetracycline-hydrocortisone oil suspension. In experimental acute trachoma the combination with hydrocortisone

seems to be more effective than tetracycline by itself. It is thought to be due to the effect of the hydrocortisone in controlling the allergic manifestations which are a feature of the acute disease. There was no advantage in the hydrocortisone-tetracycline combination in the treatment of chronic trachoma.

The author had one patient in whom the tetracycline-hydrocortisone combination made a shallow disciform central corneal ulcer very much worse, so that the ulcer became deeper and deeper and finally perforated. This complication has been noted by many other authors and emphasizes that tetracycline-hydrocortisone combinations should only be used under skilled ophthalmic supervision. *D. P. Choyce*

BLAGOJEVIC, M. [**Trachoma Control in Serbia and in Vojvodina from 1948 to 1957**] *Bull. Inst. Hyg.* Belgrade. 1959, v. 8, Nos. 1/2, 57–64. [In Serbian.]

The English summary appended to the paper is as follows:—

"In this paper the organization and the campaign against trachoma in Serbia are described. In 1948 the examination started of the whole population in endemic areas with trachoma. From 1954 on, this campaign was carried out with the help and collaboration of WHO and UNICEF. In this campaign 1,644,219 persons were examined 50,555 of whom were found to be affected with trachoma (3.07% out of the whole population). In 215 children's homes mostly out of endemic areas with trachoma 20,618 children were examined and 594 of them were found to suffer from trachoma.

"A number of institutions for trachoma treatment were founded (10 dispensaries and 180 village stations) while children from children's homes were isolated and treated in separate homes. The treatment was carried out mostly by local application of antibiotic unguents.

"The author presents the decrease in number of trachoma patients from year to year, pointing out not only the decrease of the number of patients but especially the change of the rate of patients in the progressive and regressive phase of the illness (Table No. 1). The number of patients decreased from 50,555 (in 1950) to 11,435 (in 1957). In 1951 the number of infected people in the progressive phase of the illness amounted to 17,146, while there were 16,435 patients in the regressive phase. At the end of 1957 there were only 2,406 progressive and 9,029 regressive patients; this fact is very important from the epidemiological standpoint."

MISCELLANEOUS DISEASES

ABRAHAMS, D. G. **An Unusual Form of Heart-Disease in West Africa. Its Relation to Endomyocardial Fibrosis.** *Lancet.* 1959, Aug. 15, 111–12.

During the last 2 years, 53 cases of an unusual form of heart disease in Africans were investigated in University College Hospital, Ibadan, Nigeria.

The condition, which was seen in children and young adults, appeared to be confined to the poorest classes; female patients showed a predominance over

males in the ratio of 2:1, which was of significance since males predominate in the general hospital population of Southern Nigeria. The assessed ages of the patients ranged from 6 to 47 years, 80% being below 30 years of age.

Symptoms comprised cough which, while usually dry, occasionally led to haemoptysis, dyspnoea and pain in the chest. About half of the patients presented with frank congestive cardiac failure.

The dominating physical signs were those of incompetence of the mitral valve, with pulmonary hypertension. They included signs of right ventricular enlargement with hypertrophy, a loud mitral systolic murmur often accompanied by a thrill, a split second sound at the base with accentuation of the pulmonary valve closure, and, in about 20% of the patients, a Graham Steell murmur. Mitral stenosis was never evident and the aortic valve was never affected. In 16 patients signs of acute carditis [? myocarditis] were also evident.

Fever, tachycardia, anaemia and a raised erythrocyte sedimentation rate were as constantly present as in acute rheumatism [though the occurrence of arthritis, chorea and rheumatic nodules is not recorded], and this resemblance was borne out by the finding of a raised titre of antistreptolysin-0 antibodies in the serum during the acute phase of the disease. (Sera from healthy blood donors of the same race and age-groups were used as controls in this estimation.)

Cardiac catheterization confirmed the presence of pulmonary hypertension, systolic pressures as high as 90 mm. Hg being demonstrated. The mechanism of this hypertension was puzzling since it seemed as much due to increased vascular resistance as to back pressure.

Arrhythmias other than extra-systoles were very rare.

No satisfactory follow-up records could be collected and the natural history of the disease remains conjectural. Autopsy, however, was obtained in 11 instances at which the disease process was seen both in its active and in its burnt-out stage. In the former fibrosis of the endocardium, sometimes spreading into the subjacent myocardium, was constantly present in the left ventricle; it began at the apex and spread up the inflow tract to involve the papillary muscles of the mitral valve. The outflow tract and the aortic valve were always spared. Thickening of the chordae tendineae was associated with thickening and incompetence of the mitral valve cusps. The right ventricle was always hypertrophied and the right auricle dilated, sometimes grossly so, while the main branches of the pulmonary artery were " enlarged ". In some younger patients the wall of the left auricle was thickened, wrinkled and pearly white, resembling the McCallum's patch of rheumatic carditis. Histologically the outstanding feature was the presence of perivascular aggregations of cells resembling Aschoff nodes, but these were seen only in 7 of the 11 hearts studied. These changes, together with areas of fibrinoid necrosis, evinced an intense diffuse endocardial and subendocardial inflammation, sparing the valves.

In the older subjects the heart showed less specific changes, there being fibrosis of the endocardium and subjacent myocardium of the left ventricle, with adherent thrombus, but no evidence of active myocarditis.

The author, impressed by the social status of the disease, by the serological findings and by the histopathological picture, suggests that the syndrome represents an unusual expression of the rheumatic disease. He notes that this series of patients showed features in common with cases of endomyocardial fibrosis described first by Bedford and Konstam in West African soldiers, and

later in Uganda by DAVIES, BALL *et al.* (1954) and WILLIAMS *et al.* [this *Bulletin*, 1954, v. 51, 839 ; 1955, v. 52, 203].

[Though the author has not seen the syndrome in Northern Nigerians, a group of clinically identical patients was shown to the abstracter in Kano by Dr. E. A. BEET in 1952.] *J. H. Walters*

FRASER, B. N. **Manifestations and Aetiology of Hypertension in the Coloured and Bantu.** *Brit. Med. J.* 1959, Mar. 21, 761–4. [13 refs.]

Reference to the literature reveals that the average blood-pressure readings are higher in the American adult Negro than in his white counterpart and there is evidence that the former suffers more often from essential hypertension, hypertensive heart disease and related disorders than does the latter. In Uganda, essential hypertension appears to be rare and in most cases in which the blood pressure is raised this is thought to be the result of a renal lesion. Doubt regarding the comparable position among South African Bantus and coloured persons led to this study.

It was found that the peak age of presentation of patients with benign hypertension, in both sexes, was over 60 but there was a tendency for females to present earlier than males. The shorter longevity of non-Europeans as compared with Europeans contributed to the impression that hypertension occurs at an earlier age in the former than in the latter. The causes of death were divided almost equally between cardiac failure, cerebral accidents and uraemia whereas among Europeans with hypertension approximately 50% of deaths are caused by cardiac failure, 25% by cerebro-vascular accidents and only 10% by uraemia. The lower incidence of deaths due to cardiac failure in non-Europeans may be due to the infrequency of coronary artery disease which together with coronary thrombosis is almost unknown in Bantus. Its incidence is higher in coloured persons in South Africa but it is not as high as in Europeans. The higher incidence of uraemia among the Bantus was attributed to the prevalence of primary renal disease.

At the Coronation Hospital, Johannesburg, during 1955 and 1956, 12% of all Bantus and coloured patients admitted had hypertension, and deaths from this cause amounted to one-quarter of all those occurring in the hospital. Although many fewer women than men were admitted to the hospital—largely because of the number of available beds—hypertension was more common among them than among men. The patients were very fully investigated and among 264 the hypertension was diagnosed on radiological, operative, necropsy or clinical grounds as primarily of renal origin in 70. Electrocardiographic studies revealed that left ventricular preponderance was present in the majority of patients, even among those in whom the hypertension was mild.

A. W. Woodruff

SCHRIRE, V. **The Racial Incidence of Heart Disease at Groote Schuur Hospital, Cape Town, during 1957.** *Central African J. of Med.* 1959, July, v. 5, No 7, 349–58. [26 refs.]

"(1) Three thousand nine hundred and fifty-four electrocardiograms of patients attending Groote Schuur Hospital in Cape Town during 1957 were analysed to determine the interracial incidence of heart disease.

"(2) Electrocardiographic evidence of cardiac infarction and coronary vascular disease was very common in the European, less common in the Coloured and rare in the Bantu.

"(3) A raised blood pressure was found in approximately one-third of the patients. Unlike coronary vascular disease, there was no disproportionate increase in the Europeans.

"(4) Hypertensive disease as assessed on electrocardiographic criteria of severity and by the height of the diastolic pressure was more severe in the Coloured than the European.

"(5) Hypertensive disease occurred at an earlier age in the Coloured than in the European. Females were affected more frequently than males, and Coloured females in particular developed hypertension at an earlier age than all others and of a far severer degree.

"(6) Rheumatic heart disease occurred frequently in all three races and appeared to be commonest in the Coloured.

"(7) Syphilitic heart disease was far commoner in the Coloured races than in the European.

"(8) Chronic cor pulmonale occurred with equal frequency in the three racial groups, but electrocardiographic evidence of severity was greater in the Coloured than the European.

"(9) Pericarditis occurred out of all proportion in the Bantu, being the second commonest disease in this racial group. It was far commoner in the Coloured than in the European.

"(10) Beri-beri and cardiac failure of unknown origin together were similarly commoner in the Bantu than in other races and commoner in the Coloured than in the European.

"(11) Congenital heart disease was of equal occurrence in all three racial groups.

"(12) The findings conform closely with those found between the years 1952–1956."

HILL, K. R. **Some Observations on Liver Disease in the West Indies.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1959, May, v. 53, No. 3, 217–30, 12 figs. (8 on 4 pls.) [62 refs.] Discussion 231–7, with 17 further refs. [RHODES, K.; BUNJÉ, H.; WILCOCKS, C.; WALTERS, J.; WOODRUFF, A. W.; HILL, K. R. (in reply); TODD, K. W.].

The main observations reported in this lecture are concerned with the pathology of veno-occlusive disease of the liver and vomiting sickness. They are based on experience gained in the Pathological Department at the University College of the West Indies. There are also short accounts of cirrhosis, carcinoma, yellow fever, marasmus and kwashiorkor.

The paper can be recommended as a lively introduction to the study of liver disease in Jamaica, suitable for students and newcomers to the island. The main facts have already been presented in numerous papers from the department, which have been abstracted in this *Bulletin* [see, for example, 1953, v. 50, 562; 1956, v. 53, 106, 371; 1958, v. 55, 205, 304]. *R. Passmore*

D'ABRERA, V. St. E. **Tropical Eosinophilia: an Aetio-Pathological Study. Part II.** *Proc. Alumni Ass., Malaya.* 1959, June, v. 12, No. 2, 53–77, figs. 13–24. [Numerous refs.]

This is a comprehensive review of this rather contentious subject and covers a wide well-worked field [for previous part, see this *Bulletin*, 1959, v. 56, 485].

The radiological mottling consists of discrete, soft, rounded shadows with ill-defined margins varying in size from a pin's head to 5 mm. in diameter, most numerous in the perihilar regions. These may be due to areas of cellular infiltration. In miliary tuberculosis the granulomas, in contrast, are formed by a necrotic core surrounded by a ring of lymphocytes, usually measuring 1 mm. in diameter, while in silicosis the dense fibrotic laminated nodules vary from 1 to 4 mm. Haemosiderin-laden macrophages or "heart failure cells" in chronic heart disease may accumulate in amounts sufficient to produce radiological mottling. Recently a condition of idiopathic pulmonary haemosiderosis has been described and is associated with repeated intrapulmonary haemorrhages. These have to be distinguished from true tropical eosinophilia.

It is uncertain whether the eosinophilia is produced by infections or by allergy. The former are inflammatory responses produced by irritants, while allergy or hypersensitivity reactions are those initiated by allergens.

7 fatal cases of tropical eosinophilia consisted of 3 adults and 4 children, and formed the basis of this study. (One has previously been described by BEAVER and DANARAJ [this *Bulletin*, 1958, v. 55, 791] as pulmonary ascariasis resembling eosinophilic lung.)

Possibly the true explanation of this syndrome is that it is produced by a variety of migratory nematodes, the predominating agent varying in different countries. In those cases due to filariae where no microfilariae can be found in the bloodstream or lymp nodes, the reactions may have resulted from the migrations of zooparasitic larvae alone or from "visceral larva migrans".

If animal-host filariae (*Dirofilaria, Setaria, Wuchereria pahangi*, etc.) could grow to maturity and produce microfilariae in man, then either these parasites are equally adaptable in man, or new conceptions will have to be made of host-parasite relationships.

[This paper contains a criticism of BUCKLEY's pioneer work on this difficult subject (this *Bulletin*, 1958, v. 55, 1356).] *Philip Manson-Bahr*

NATH, K. & PANDEYE, S. N. **Aberrant Presentations of Tropical Eosinophilia.** *J. Ass. Physicians India.* 1959, July, v. 7, No. 4, 310–19, 2 charts. [35 refs.]

A series of 8 atypical cases of tropical eosinophilia which have been investigated during the last 2 years is discussed.

All the patients were Indian males ranging from 18 to 75 years of age. They presented various dominant features, such as continued pyrexia and generalized lymphadenitis, dry pleurisy, pleurisy with effusion, bronchial asthma, gastro-intestinal disorders, tender liver, and papular dermatitis. Terms like "pseudotuberculosis", "pulmonary eosinophilosis", "benign eosinophilia", and "tropical pulmonary eosinophilia", used by various authors, show that pulmonary manifestations along with eosinophilic leucocytosis have been the chief criteria for the diagnosis of tropical eosinophilia.

The only common feature in all the present cases was indeed an eosinophilic leucocytosis and the dramatic response to diethylcarbamazine therapy.

From a study of the literature it appears that renal involvement (nephritis), and cardiac manifestations have been described respectively by MEYERS and KOUWENAAR [this *Bulletin*, 1939, v. 36, 838] and by D'ABRERA [*ibid.*, 1958, v. 55, 1051], but generalized lymphadenopathy constitutes a new manifestation.

In the light of aberrant presentation and extra-pulmonary involvement the term " tropical pulmonary eosinophilia " should be discarded and the older original " tropical eosinophilia " substituted for it.

Philip Manson-Bahr

BAKER, S. J., RAJAN, K. T. & DEVADATTA, S. **Treatment of Tropical Eosinophilia. A Controlled Trial.** *Lancet*. 1959, Aug. 22, 144–7, 5 figs. [11 refs.]

A considerable series—165 cases—of tropical eosinophilia (all in Indians) was studied. Of these patients 43% were under 10 years of age and 24% had hepatomegaly.

A controlled blind trial with placebo, Acetylarsan and diethylcarbamazine was carried out in 75 patients. They were divided into 3 groups, as follows.

Group A received placebo tablets and placebo injections. The tablets were indistinguishable from diethylcarbamazine and the injections indistinguishable from Acetylarsan injections.

Group B received placebo tablets and injections of genuine Acetylarsan.

Group C received genuine diethylcarbamazine tablets and placebo injections.

Tablets were given for 1 week and injections twice weekly for 5 weeks.

The dose of tablets was 10 mgm./kgm. daily and intramuscular injections were according to the following schedule:

for children under 20 kgm. weight, 0·5 ml. for the first injection, 0·75 ml. for the second and then 1 ml. twice a week ; for children over 20 kgm., 0·5 ml., 0·75 ml., 1 ml. and then 2 ml. twice a week. The preparation containing genuine arsenic for children had 0·02 gm. arsenic per ml.

Adults received 0·5 ml. for the first injection, 1 ml. for the second and 2 ml. thereafter: the preparation of arsenic contained 0·05 gm. per ml.

A patient was considered cured when symptoms and signs had disappeared and the eosinophil count was less than 2,000 per cmm. In each case, after completion of 5 weeks' course of treatment, observation of the cured patients was continued for 2 months. The majority belonged to the poorer classes from the town of Vellore and surrounding rural areas. The preponderance of males over females was in the ratio of 3:2. Some clinical details are given.

Of the 25 patients in group A (placebo tablets and placebo injections) none improved spontaneously. Of those in group B (placebo tablets and Acetylarsan injections) 15 responded, but 7 of these relapsed subsequently. Of the 25 in group C (diethylcarbamazine tablets and placebo injections) 24 responded and the response in general was quicker than in those given arsenic.

The results of a trial with varied doses of diethylcarbamazine suggested that 5 mgm. per kgm. per day, for 3 days is as effective as 10 mgm. per kgm. given for a week. This dosage level is considerably less than that recommended

by DANARAJ in Singapore [this *Bulletin*, 1958, v. 55, 920] (18–30 mgm. per kgm.). The ready and rapid response is further evidence that the disease may be caused by some form of filaria. *Philip Manson-Bahr*

i. SIMPSON, D. A., LANDER, H. & ROBSON, H. N. **Observations on Kuru: II. Clinical Features.** *Australasian Ann. of Med.* 1959, Feb., v. 8, No. 1, 8–15.

ii. FOWLER, M. & ROBERTSON, E. G. **III. Pathological Features in Five Cases.** *Ibid.*, 16–26, 17 figs. [17 refs.]

i. The neurological findings are recorded in detail of 27 patients, 18 females and 9 males of ages ranging from 6 to 40 years, suffering from kuru. These patients had been investigated previously by ZIGAS and GAJDUSEK [this *Bulletin*, 1958, v. 55, 1048] in the Fore region of Eastern New Guinea. The authors found evidence of widespread involvement of the central nervous system, but predominantly evidence of progressive cerebellar dysfunction associated in most with alterations in tone and involuntary movements. "Kuru tremor", seen in 23 of the patients, is a fine regular tremor, like shivering, involving muscles carrying out sustained contraction as in maintaining posture. The authors suggest that oscillating discharge of the spinal motor neurons, potentiated by voluntary innervation, provides an explanation of this. Other involuntary movements, more typical of involvement of the extrapyramidal system, were seen in a few patients. While there was some increase in tendon reflex responses these were unaccompanied by weakness, spasticity, or constant extensor plantar responses, so that there was only minimal evidence of involvement of the pyramidal tracts. The assessment of intellectual function and emotional reaction is difficult for investigators not fluent in the language of their patients; but the authors noticed pathological smiling and laughing, and believed that advanced cases of the disease showed some dementia. Their impression was that the speed of progression of the disease varied from time to time in individual patients, but there is no good evidence of natural cure. Those patients who were said to have recovered from kuru were thought by the authors probably to have been cases of hysterical mimicry.

ii. This paper records in detail the histological changes found on examination of 5 brains and 2 spinal cords from patients who had died of kuru. In 2 cases some clinical data were available, and in these the brains and spinal cords, as well as tissues from other sites were examined; but in 3 cases no clinical details are available and only the brains could be examined. Diffuse degenerative changes were found and there is evidence of a wide spectrum of neuronal susceptibility. The most conspicuous change was degeneration of the cell bodies which showed, in most cases, various degrees of shrinkage; but in many neurons there was also vacuolation of the cytoplasm. The cells of the cerebellar cortex, the dentate nucleus, thalamus, corpus striatum, globus pallidus, and some areas of the cerebral cortex showed these degenerative changes, associated with some gliosis. Both the spinal cords showed demyelination in the lateral cortico-spinal tracts, in one throughout the cord, in the other most marked in the lumbo-sacral region. One of these patients exhibited during life brisk knee jerks, ankle clonus, and absence of both

lower abdominal reflexes, but the plantar responses were flexor. Details of the reflexes in the other patient are not available.

The authors summarize their observations in relation to other diseases affecting the central nervous system, pointing out the absence of evidence of an inflammatory response, or of signs of virus infection. Superficial resemblances to manganese poisoning, and to some features of organic mercury poisoning are mentioned. The probability of avitaminoses occurring late in the disease is indicated by pathological evidence in one of these cases of Wernicke's encephalopathy ; but there is no evidence that vitamin deficiencies play a causative role. The closest affinities of the disease are to the group of hereditary spino-cerebellar degenerations. *S. Bell*

PARASITOLOGY: GENERAL

MALDONADO, J. F. & OLIVER-GONZALEZ, J. **The Prevalence of Intestinal Parasitism in Certain Areas of Puerto Rico—a 3-Year Study.** *Bol. Asoc. Med. de Puerto Rico.* 1959, Apr., v. 51, No. 4, 109–24. [22 refs.]

The prevalence of intestinal parasites in the population of Puerto Rico has been studied intermittently for some 30 years [this *Bulletin,* 1928, v. 25, 244 ; 1931, v. 28, 665 ; 1935, v. 32, 244 ; 1946, v. 43, 1155]. The survey reported here differs from earlier ones in that it was repeated annually 3 times in succession, and that a large number (23,262) of samples of faeces were examined from 6 localities representing the 3 physiographic areas of the island. Specimens were obtained from approximately equal numbers of the sexes, but about 70% of the donors were children under 15 years. The specimens were examined by direct and concentration methods. The findings indicate very little improvement in the sanitary habits of the population.

Ascariasis remains an important problem. The peak incidence was reached between 5 and 10 years of age and then tended to decrease. Among nearly 1,900 egg-counts 14·5% had more than 20,000 eggs per gm. of faeces. 40% of persons showed only infertile eggs and this was more common in areas of low prevalence. *Trichuris trichiura* was the commonest helminth with an incidence ranging from 76% to 88%. Persons under 15 showed the highest rate but more than 60% of those over 41 years were parasitized. The infections were light. Hookworm incidence was 30% or higher except in the dry coastal zone. It reached its peak incidence between 11 and 25 years, but the incidence was lower than in earlier surveys. Only 2% carried a worm load of over 400 and the average load was 60 to 70 worms. *Strongyloides* infection was present only in 5%. This parasite has never been reported as common, but survey methods were not really suitable for its diagnosis.

Infection with intestinal protozoa differed little from the situation revealed previously. *Entamoeba histolytica* cysts were found in 9·2% to 17·7% of specimens in different years. The high incidence in women of reproductive age suggested the possibility of transmission to their children. *Entamoeba coli* was the most common protozoon and, like all the other protozoa except *Giardia intestinalis,* was significantly more prevalent in women. *Endolimax nana* was

the next commonest infection, and *Iodamoeba williamsi* was the least common. *G. intestinalis,* the only flagellate common in this survey, was more prevalent in the warm, dry areas at lower altitudes. The incidence was highest in children and decreased with age. *T. H. Davey*

ENTOMOLOGY AND INSECTICIDES: GENERAL ZOOLOGY

[Papers on the toxic effects of insecticides in man are abstracted in the *Bulletin of Hygiene* under the general heading of Occupational Hygiene and Toxicology.]

COLLESS, D. H. **Notes on the Culicine Mosquitoes of Singapore. V.—The *Aëdes niveus* Subgroup (Diptera, Culicidae): Previously Described Species and Keys to Adults and Larvae.** *Ann. Trop. Med. & Parasit.* 1959, June, v. 53, No. 2, 166–79. [10 refs.]

See this *Bulletin,* 1959, v. 56, 669.

GILLETT, J. D., HADDOW, A. J. & CORBET, P. S. **Observations on the Oviposition-Cycle of *Aëdes* (*Stegomyia*) *aegypti* (Linnaeus), II.** *Ann. Trop. Med. & Parasit.* 1959, Apr., v. 53, No. 1, 35–41, 1 fig.

There is a daily cycle of oviposition in *Aëdes aegypti,* the main peak in the afternoon, under natural conditions. It persists if artificial lighting is substituted for the daylight period, even with abrupt change from light to dark. These facts refer to alternating 12-hour periods of light and dark, approximating natural timings in equatorial regions. The oviposition rhythm persists in the same relation to light and dark, if these are reversed. Constant light or constant dark results in a loss of the rhythm. It is, however, still apparent with only 4 hours of light or 4 hours of darkness, in the 24-hour schedule, although less so than if these times are increased. In discussion, it is concluded that, probably, the oviposition cycle involves an endogenous rhythm, innate or acquired, but it is actuated by time-cues in terms of light and dark.

[See also this *Bulletin,* 1958, v. 55, 106.] *D. S. Bertram*

MCKIEL, J. A. **Sensitization to Mosquito Bites.** *Canadian J. Zool.* 1959, June, v. 37, No. 3, 341–51. [19 refs.]

Economic developments in Canada and the country's strategic position have led to studies on the control of biting insects. It is a serious problem, despite available means of control and prevention. This paper is a study of bite reactions as part of a general programme against biting pests.

No difficulty was experienced, as by earlier workers [this *Bulletin,* 1949, v. 46, 514], in the present work in inducing sensitization of rabbits and guineapigs (and white rats) to the bites of mosquitoes. Several species were available for use (*Aëdes aegypti, Aëdes atropalpus, Aëdes vexans, Culex restuans, C. pipiens* and some others). For rabbits, 2 hours' exposure to large numbers of mosquito bites for 5 days, and 5 days' continuous exposure for guineapigs and

white rats, sufficed to sensitize them. Also, serial injections of mosquito extracts (*Aëdes aegypti*) were effective. Later, two bites per day were also found satisfactory and most consistent in producing sensitization. In sensitized animals, the immediate weal reaction appears after 15 minutes and is maximal in an hour, that is, slower than in man. Erythema is not marked. The weal may disappear in 8 hours or persist in papular form for 2 or 3 days. Delayed reactions in response to bites, certainly separate from the weal reaction, were not obtained in the animal material. Some work is here done on it in human beings.

In discussion, it is concluded that both reactions are of allergic nature and not due to a toxin injected during biting. No support is given to the concept of histamine being injected by the mosquito and being causative of reactions. The antigenic material is thought to be more complex. It is located, certainly in the head-thorax region, presumably in the salivary glands. Injected material from the mosquito's abdomen was not fully antigenic, as this did not sensitize normal animals. *D. S. Bertram*

LEWIS, D. J. & GARNHAM, P. C. C. **The Species of *Phlebotomus* (Diptera: Psychodidae) in British Honduras.** *Proc. Roy. Entom. Soc. of London.* Ser. B. 1959, June 19, v. 28, Pts. 5/6, 79–89, 50 figs. [19 refs.]

14 species of *Phlebotomus* from British Honduras were recorded during early 1958. Taxonomic details and notes on distribution are given. A description of a possible male of *P. permirus* is given and of the female of *P. ovallesi*.

[See also this *Bulletin*, 1959, v. 56, 930.] *E. R. Nye*

LABRECQUE, G. C., BOWMAN, M. C., ACREE, F., Jr. & SMITH, C. N. **Adsorption of DDT and its Absorption and Conversion to DDE by House Flies exposed to Residues.** *J. Econom. Entom.* 1959, June, v. 52, No. 3, 528–9.

A very simple method of testing for resistance in house-flies is to expose them to dry residues of insecticide in glass jars and determine the knock-down times. This method seems to work fairly well in tests of resistance to BHC and dieldrin, but anomalous results were obtained with DDT.

It was decided to investigate the relations between the doses picked up by flies crawling over treated glass, and the rate of deposit and exposure period. Flies from resistant and normal colonies were confined in 1-pint glass jars treated with volatile solutions of DDT to leave deposits of 2 or 3·5 mgm. per square foot of DDT. At intervals, batches of flies (crawling or paralysed) were removed and analysed for DDT externally and for DDT and DDE internally.

The results showed that the dose picked up from 2 mgm./sq. ft. by one colony was very roughly in proportion to that acquired from 3·5 mgm./sq. ft. by another colony. But other results were anomalous. In some colonies there was steadily increasing dose with exposure time; in others the rate of pick-up seemed to fall off or even decline after a certain time. Even more striking was the difference in doses picked up by the various strains. The following

extract from the results illustrates this. (Data for non-paralysed flies after 30 minutes' exposure.)

Colony	μgm. per 100 flies External DDT	Internal DDT	Internal DDE	Total
Normal	29·2	7·3	0	36·5
Slightly resistant	17·2	2·4	2·7	22·3
Highly resistant	6·0	·7	1·8	8·5
Malathion resistant	8·1	2·6	2·3	13·0

(The malathion resistant colony, selected by malathion from the normal strain, had appreciable DDT resistance, though never exposed to DDT.)

One possible explanation for the differences in pick-up was that normal flies were much more active than resistant ones. [Possibly this was due to an irritant effect of DDT not affecting the resistant flies.] *J. R. Busvine*

JARMAN, R. T. **The Deposition of Airborne Droplets on Dead House-Flies (*Musca domestica* L.).** *Bull. Entom. Res.* 1959, Aug., v. 50, Pt. 2, 327–32, 1 fig. [14 refs.]

"An experimental laboratory study of the deposition of droplets on dead house-flies (*Musca domestica* L.) was made, using a spinning-top sprayer to produce a spray of uniformly sized oil droplets and a cascade impactor to measure the concentration of the spray of droplets, which were dyed. The deposits obtained on a dead house-fly and a cascade-impactor slide when these were exposed in turn to a wind of 1 m. per sec. in a wind tunnel were compared colorimetrically, and determinations thus made of the collection efficiency of the flies, defined as the volume of liquid deposited on an object expressed as a percentage of the volume that would have passed through the same cross-section had the object not been there.

"The measured collecting efficiency of a fly varied from about 70 per cent. (droplet dia. 27μ) to about 200 per cent. (droplet dia. 75μ), and was approximately twice that of a sphere with a cross-sectional area twice the projected frontal area of the fly. From theoretical calculations of the filtering effect of different elements of the vegetation, it is concluded that the optimum droplet diameter for deposition on flies in woodland is 20–40μ."

O'BRIEN, R. D. & WOLFE, L. S. **The Metabolism of Co-Ral (Bayer 21/199) by Tissues of the House Fly, Cattle Grub, Ox, Rat, and Mouse.** *J. Econom. Entom.* 1959, Aug., v. 52, No. 4, 692–5, 5 figs. [16 refs.]

KANO, R. **Notes on Flies of Medical Importance in Japan. Part XIV. Descriptions of Five Species belonging to Chrysomyinae (Calliphoridae) including one Newly Found Species.** *Bull. Tokyo Med. & Dental Univ.* 1958, Sept., v. 5, No. 3, 465–74, 1 map, 1 fig. & 4 pls. [15 refs.]

DIAS, J. A. T. S. Contribuição para o estudo dos tabanídeos (Diptera, Tabanidae) de Angola. [**Study of Tabanids in Angola**] *Anais Inst. Med. Trop.* Lisbon. 1958, Sept., v. 15, No. 3, 781–824, 4 figs. & 1 map. [13 refs.]

Two new species from the Ethiopean Region are described, *Tabanus sousadiasi* (with a key to the subgenus *Hybomitra*) and *Haematopota machadoi* (near *H. duttoni*), and 65 species of Tabanid from Angola are listed with locality records. Most of the species recorded are equally divided between the genera *Tabanus* (*sensu lato*) and *Haematopota*, and only 5 species of the subfamily Pangoniinae, including 3 species of *Chrysops*, have been found in this area.

B. R. Laurence

MOHR, C. O. & MORLAN, H. B. **The Nature of Parasitism of the Opossum by Fleas in Southwestern Georgia.** *J. Parasitology.* 1959, Apr., v. 45, No. 2, 233–7.

The opossum, *Didelphys marsupialis*, has very few specific ectoparasites although it harbours temporarily triatomid bugs, ticks, mites and fleas of several other animals, probably because it sometimes nests in their abandoned dens and it frequents a variety of environments—fields, light woodland, roadsides, creeks, treeholes and even nests in barns and sheds. This paper reports on fleas from 349 opossums examined in south-western Georgia from 1945 to 1949, in connexion with murine typhus investigations; they were caught in the field and in buildings in DDT-treated and in untreated areas. The following fleas were rarely or infrequently encountered: *Pulex irritans*, or possibly *P. simulans* [this *Bulletin*, 1959, v. 56, 438]; species of rabbit fleas; *Leptopsylla segnis*; *Xenopsylla cheopis*; the stick-tight flea of poultry. Percentages of opossums infested were 9% or usually less, and the number of fleas per animal usually only 1 or 2. Squirrel fleas were rather more frequently encountered on the whole. Seasonal occurrence only was notable for some of these flea species. On the other hand, *Ctenocephalides felis* and the cotton rat flea (*Polygenis gwyni*) occurred in 11 to 12 months of the year, mean percentage infestations being, respectively, nearly 40% and a little under 30%. Heavy individual infestations occurred. Seasonal differences were notable.

Cat, stick-tight and rat fleas were commoner on opossums from occupied farms than on specimens taken in the open or vacated steadings. DDT treatment had little effect on the flea infestations on the opossum. *D. S. Bertram*

MCGINNISS, G. F. **Avian-Mite Dermatitis. Report of a Case.** *New England J. of Med.* 1959, Aug. 20, v. 261, No. 8, 396–7, 1 fig.

"A case of severe pruritus due to infestation by mites identified as *Ornithonyssus sylviarum*, the Northern fowl mite, is presented. The mites came from infested birds' nests, which had been built in an unscreened air-conditioning unit. Mite infestation should be considered when pruritus is encountered in patients using air conditioners."

[See this *Bulletin*, 1958, v. 55, 1181.]

HOOGSTRAAL, H. & THEILER, Gertrud. **Ticks (Ixodoidea, Ixodidae) parasitizing Lower Primates in Africa, Zanzibar, and Madagascar.** *J. Parasitology.* 1959, Apr., v. 45, No. 2, 217–22. [11 refs.]

In view of interest in piroplasms and relapsing fever spirochaetes known from African monkeys, the ixodid tick records for primates—other than man—in the African mainland, Zanzibar and Madagascar are brought together in this paper. Much hitherto unpublished data by the authors and colleagues are included. The following ixodids were taken from pottos, galagoes, bushbabies, lemurs, monkeys (*Cercopithecus* and *Colobus*) and baboon: *Amblyomma hebraeum*, *A. variegatum*, *Haemaphysalis l. leachi*, *H. parmata*, *Hyalomma truncatum*, *Ixodes cavipalpis*, *Rhipicephalus appendiculatus*, *R. e. evertsi*, *R. pravus*, *R. s. sanguineus*, and *R. s. simus*, as well as ticks known to be host-specific to primates, *viz. Haemaphysalis lemuris*, *I. lemuris* and *I. schillingsi*. Argasids have not yet been recorded as feeding on lower primates in nature.

It is commented that there is much scope for study on the relation of these tick parasites to the transmission of the pathogens of lower primates.

D. S. Bertram

HEISCH, R. B. & MCPHEE, R. **Notes on Some Ixodid Ticks in the Nairobi Area.** *East African Med. J.* 1959, Apr., v. 36, No. 4, 187–99, 12 figs.

Around Nairobi 3 species of hard tick, *Haemaphysalis leachi, Rhipicephalus simus* and *Amblyomma variegatum*, have been shown to carry infections of the *Rickettsia conori* type [this *Bulletin*, 1957, v. 54, 1068 ; 1958, v. 55, 144]. In addition, the first two species have been found in rodent burrows, and the rodents, *Arvicanthis*, *Otomys* and *Lemniscomys*, are also reservoirs of the infection. As it is important in this endemic area that the species of tick found biting man should be identified, the authors have compiled a key, with brief descriptions and host list, to the 10 common species of *Haemaphysalis*, *Rhipicephalus*, *Amblyomma* and *Boophilus* found here. [One would like to know more about the authors' "academic entomologist" who might object to brief descriptions of species and simplified keys. Either the descriptions and keys are adequate, or they are inadequate. Those given here appear to be adequate for the purpose of identifying the 10 species mentioned. One of the pleasing features of the paper is that both sexes of all the species are figured.]

B. R. Laurence

HOOGSTRAAL, H. **Biological Observations on certain Turkish Haemaphysalis Ticks (Ixodoidea, Ixodidae).** *J. Parasitology.* 1959, Apr., v. 45, No. 2, 227–32, 1 fig.

This paper brings together data available, outside the U.S.S.R., on haemaphysalid ticks of Turkey, combining collection records of NAMRU-3 in the autumn of 1954 and examinations of material in the British Museum (Natural History) and the Rocky Mountain Laboratory. The species and forms are: *Haemaphysalis concinna*, *H. erinacei erinacei*, *H. e. taurica*, *H. e. turanica*, *H. otophila*, *H. punctata*, *H. sulcata* and *H. inermis inermis*. No specimens were found satisfying a description of *H. angorensis* of questionable validity. The hosts of the adults are variously cattle, sheep, goat and camel. NAMRU-3

catches of 400 small mammals now add considerably to host information for immature stages, larvae and nymphs being taken on a range of rodents and the hedgehog, birds and lizards. The tick-infested hosts were those found in steppeland, fields, pastures, gardens, stunted oak forest, never in humid forest. The significance of further study on their host relationships is emphasized in relation to vector functions, a topic which is briefly commented upon for each tick. *D. S. Bertram*

HOOGSTRAAL, H. & KAISER, M. N. **The Ticks (Ixodoidea) of Iraq: Keys, Hosts, and Distribution.** Reprinted from *J. Iraqi Med. Professions.* 1958, June–Sept., v. 6, Nos. 2/3, 22 pp., 1 map on pl. [10 refs.]

The authors studied several collections of ixodid ticks from Iraq and report on them in this paper. The number of species is shown in brackets for the several genera concerned: *Argas* (3); *Ornithodorus* (3, probably more); *Boophilus* (2); *Haemaphysalis* (4); *Hyalomma* (6); *Ixodes* (a new species being described elsewhere, and indeterminate spp.); *Rhipicephalus* (2). The list, it is noted, is certainly incomplete and, moreover, is based chiefly on river valley collections. Some indication is given of useful lines for further surveys and work. Records for each species give known details of distribution, hosts and tick stages on the hosts. A separate list names tick species on each host. Keys are provided to genera and species, including hitherto confusing *Hyalomma* species. *R. sanguineus secundus* is only recognizable to the authors in Palestine material, so that only *R. sanguineus sanguineus* is recognized by them as present in this Iraq material. The only illustration is a map of localities. *D. S. Bertram*

COLE, M. M., LABRECQUE, G. C. & BURDEN, G. S. **Effects of Gamma Radiation on some Insects affecting Man.** *J. Econom. Entom.* 1959, June, v. 52, No. 3, 448–50.

"The LD-50 of gamma radiation from cobalt-60 against the body louse (*Pediculus humanus humanus* L.), house fly (*Musca domestica* L.), American cockroach (*Periplaneta americana* (L.)), German cockroach (*Blattella germanica* (L.)), firebrat (*Thermobia domestica* (Pack.)), bed bug (*Cimex lectularius* L.), and Pharaoh ant (*Monomorium pharaonis* (L.)) ranged from 130 r for half-day-old house fly eggs to 190,000 r for body louse nymphs and Pharaoh ant queens. Doses required to cause 100% mortality ranged from 600 r to 250,000 r. Among the species tested, the LD-50 varied inversely with the size of the insect. Reproduction in body lice was inhibited at dosages of 75,000 r or higher. DDT-resistant body lice were as susceptible to gamma rays as non-DDT-resistant lice."

REID, J. A. & CHEE SINN LIM. **A Note on the Residual Life of the Insecticide Malathion.** *Med. J. Malaya.* 1959, Mar., v. 13, No. 3, 239–42, 1 fig.

With increasing numbers of anopheline species showing resistance to control by the chlorinated hydrocarbon insecticides, attention is turning to organophosphorus compounds as possible alternatives. One of the most interesting

is malathion, which is known to be very safe, as its toxicity to mammals seems to be less than that of DDT. The main objection seems a rather short residual life. KRISHNAN *et al.* [this *Bulletin*, 1959, v. 56, 190] treated houses in Indian villages at the rate of 25 mgm./sq. ft. and concluded, from subsequent mosquito densities in the houses, that its insecticidal powers were lost in 3 weeks. (On the other hand, DDT at 200 mgm./sq. ft. lasted effectively only 5 weeks.) LABRECQUE *et al.* [*ibid.*, 1958, v. 55, 1303] made laboratory trials by spraying plywood panels and exposing *Anopheles quadrimaculatus* (for periods ranging from 5 to 120 minutes) at intervals afterwards. In these tests they found that both malathion and DDT were still effective after 6 months, judging by the longer exposure results.

The present authors have sprayed an old creosoted wooden wall with doses of 50 and 100 mgm./sq. ft. malathion and tested for residual action at intervals by confining *Aëdes aegypti* against the wall for 5-, 30- or 60-minute periods. Basing their results on the kills 24 hours after a 1-hour exposure, they consider that the insecticidal action remains effective (above 60% kill) for 3 months and falls sharply thereafter, all being lost by 6 months. It was not possible to make valid comparisons with DDT, because the local strain of *Aëdes aegypti* was found to be resistant to this insecticide, by the Busvine-Nash test.

[These results and those of LaBreque *et al.* are rather favourable to malathion by the somewhat long exposure period. Nevertheless, they seem encouraging.] *J. R. Busvine*

COURSHEE, R. J. **Drift Spraying for Vegetation Baiting.** *Bull. Entom. Res.* 1959, Aug., v. 50, Pt. 2, 355–70, 14 figs. [19 refs.]

UNGUREANU, E. & UNGUREANU, L. Une micropipette pour l'application topique de très petites quantités d'insecticide. [**A Micropipette for Topical Applications of Very Small Quantities of Insecticide**] *Bull. World Health Organization.* Geneva. 1959, v. 20, No. 5, 1005–7, 1 fig.

A micropipette for the application of minute quantities of insecticide to insects is described. The apparatus consists of a capillary tube 0·02–0·03 mm. in internal diameter, closed at its upper end by a piece of rubber tubing 3 cm. long, into which is inserted a short piece of glass rod. A cotton or nylon thread is attached to the upper part of the rod and to a screw on the support; turning the screw winds in the thread which in turn bends the piece of rubber tubing; the small change in volume produced in the rubber tubing is transmitted to the capillary tubing. Instructions are given for drawing the pointed delivery end of the tube and the calibration of the apparatus. *E. R. Nye*

LABORATORY PROCEDURES

McArthur, J. **A New Concept in Microscope Design for Tropical Medicine.** *Amer. J. Trop. Med. & Hyg.* 1958, July, v. 7, No. 4, 382–5, 4 figs.

The author gives an account of a most ingenious and effective microscope which he invented 25 years ago and improved while he was a prisoner of war in Japanese hands. Since then the microscope has been produced for sale on a continually increasing scale, and the later models incorporate several new features.

The most immediately obvious advantages of this microscope are that it is small (4 × 2½ × 2 inches, easily fitting into the hand), light (18 ounces), rigid, and independent of any source of light other than its own built-in source provided by electric batteries. It can therefore be used anywhere—in the laboratory, in an aircraft or, for microphotography, on top of a London bus (as an illustration proves). But it has other advantages. The slide to be examined is placed face downwards on the stage, and the 3 objectives are fixed beneath the stage, directed upwards, and automatically in focus; only fine adjustment is necessary, and there is no racking up and down as in conventional models. Light is directed to the eyepiece by prisms, and the image is right side up. The objectives are conventional low and high power and oil-immersion, but other models are available. Various additional pieces of equipment, such as binocular eye-pieces and attachments for photography, can be obtained.

The microscope will do everything that a conventional instrument will do, and possesses many real advantages. [It is a most stimulating example of human ingenuity and skill, and it is beautifully made.] It is already finding great favour among scientists who work in difficult field conditions.

Charles Wilcocks

MISCELLANEOUS PAPERS

Anais Inst. Med. Trop. Lisbon. 1958, Sept., v. 15, Suppl. No. 2, 3–683, numerous illustrations. **Número especial dedicado aos VI Congressos Internacionais de Medicine Tropical et de Paludismo.**

A special number of this journal was prepared for the 6th International Congresses of Tropical Medicine and Malaria and it deals with the health services in Portuguese territories overseas and with contributions of Portuguese doctors and others to tropical medicine through the ages. The first two papers describe the organization and functioning of overseas health services, which were reorganized in 1945 when they ceased to be military and became civilian in character. The aims of the health services are discussed, the problems encountered, the needs of the territories and the results achieved.

Then follow 8 papers written by the directors of the health services in the overseas territories, reporting on the particular territories in their charge, namely Cape Verde, Portuguese Guinea, San Thome and Principe, Angola, Mozambique, Goa, Macau and Timor. There is also an account of the work

of the Tsetse and Trypanosomiasis Department in Mozambique. It is clear that since the reorganization of the services much more interest has been taken in hygiene and preventive medicine. Intensive attacks are being made on malaria, leprosy, filariasis and tuberculosis in all territories, and on yaws and sleeping sickness in African territories.

The final chapter gives a historical outline of Portuguese contributions to tropical medicine, starting in 1147 when the Moors were defeated. Though the Portuguese navigators had very many contacts with tropical countries they did not publish their experiences. There were, however, some notable contributions, such as ORTAS's (1563) book on Indian medicines and Aleixo de ABREU's volume on the diseases of hot countries in 1623. During the 16th and early 17th centuries an overseas hospital system of at least 40 hospitals was built by the Portuguese in Africa and Asia. In 1691 two doctors were sent to Goa to teach medicine and surgery, an early precursor of the modern schools of medicine in the tropics. Other interesting dates are 1844 when 5 overseas medical schools were established and a school for "the instruction of native health officials", and 1859 when a Department of Naval and Overseas Health was established in Portugal. A Chair in Tropical Medicine in Portugal was proposed in 1883 but not founded, and in 1902 a hospital for the treatment of the sick from overseas was opened. The School of Tropical Medicine in Lisbon was founded in the same year. Many references are given and one is impressed by the large amount of work done by Portugese workers in the field of tropical medicine.

[See also this *Bulletin,* 1953, v. 50, 355.] *T. H. Davey*

VYAS, K. J. **Liver Palpability in Normal Infants and Children.** *Indian J. Child Health.* 1959, Aug., v. 8, No. 8, 439–41.

"715 healthy infants and children were examined to study the normal liver palpability. The liver was found palpable in 155 children, the highest figure being in the age group of 4–6 months. The extent of palpability varied from 1½ to 9 Cms., the commonest figure being 3 Cms."

INTERNAT. NURSING REV. London. 1958, Oct., v. 5, No. 4, Suppl., 31 pp. **The Essentials of Tropical Disease Nursing.**

This Supplement to the *International Nursing Review* is a paper prepared by the Trained Nurses Association of India as one of a series written by the nurses of different countries on behalf of the Nursing Service Committee of the International Council of Nurses. The intention of this series was to give some indication of the standards of nursing which are required in the case of patients suffering from different types of illness. This particular paper goes further than this. It is the report of a study of tropical disease nursing and has outlined the modern concept of tropical diseases, the classification of these diseases, trends in their control and some indication of the international and national measures which have been or should be, in the opinion of the Trained Nurses Association of India, included in organized programmes for the prevention and management of these diseases. All this, as the paper says, has been set out to facilitate understanding of the significance and implications of nursing in these organized programmes. The paper then gives a clear and

concise description of the nursing needs of patients suffering from most of the tropical diseases.

Certain of the recommendations made and of the examples given in regard to programmes for the control of tropical diseases obviously relate to India and might not be applicable to the same extent in other countries, but this does not seriously detract from the value of the paper, which could be studied with benefit by all those interested in or concerned with the nursing of patients in the tropics, whether or not these patients are suffering from the conditions usually included under the heading of tropical diseases.

F. N. Udell

REPORTS AND SURVEYS

EAST AFRICA HIGH COMMISSION. **East African Institute for Medical Research Report, 1957–1958** [HOLMES, E. G., Director]. 29 pp., 1 fig. & 1 map. 1959. Nairobi: Govt. Printer. [Sh. 2.]

The main interest of research in this Institute continues to lie in the study of body composition, body fluid balance, and other fundamental biochemical studies. There has been much concentration on the use of radio-isotopes in the study of the distribution of water throughout the body and the following have been or are being used: ^{3}H, ^{14}C, ^{85}Kr, ^{82}Br, ^{22}Na, ^{90}Sr. The technique by which they are used is outlined, together with some findings which include the establishment of a satisfactory method of determining total body water by the tritrium dilution method, demonstration that changes in body weight do not necessarily parallel those in body solids, and elaboration of the technique for the study of extracellular body fluid by sodium thiocyanate, while progress has been made towards an independent method for the measurement of the body fat which is badly needed for the study of cell hydration. There have also been considerable studies on the degree of absorption of nutrients from foodstuffs. It has been shown that this is lower than is implied by the factors 4, 9 and 4 for the calorie values of carbohydrate, fat, and protein which are normally used, and the present studies relate to factors which might have influenced absorption. They have been paralleled by studies on the loss of nitrogen from the skin which is shown to be material. There is an interesting study of nutritional ophthalmology in which it is suggested that nearly half the total of cases of blindness are attributable to keratomalacia, trachoma and spontaneous iris prolapse which have a direct or indirect relationship to malnutrition.

Previous studies of the possibility of controlling filariasis by mass chemotherapy continue with still promising but not yet completed results. The geographical distribution of infections with *Schistosoma haematobium* and *S. mansoni* on the southern shores of Lake Victoria has been mapped and the relative incidence suggested that infection with the latter comes from the lake, though that of *S. haematobium* cannot yet be explained. Studies of the carrier

have been somewhat more fruitful than in past years and there have been a few findings of infected snails, probably *Bulinus nasutus*.

The report also includes a brief note on the 6 biological variants of *Ornithodorus moubata* which differ in their distribution and their host preferences and thus probably in their relationship to the epidemiology of relapsing fever.

G. Macdonald

McCulloch, Merran. **A Social Survey of the African Population of Livingstone.** pp. x + 82, 3 figs. & 1 folding map. 1956. Manchester 13: Manchester University Press, 316–324, Oxford Road. [For Rhodes-Livingstone Institute.] [10s. 6d.]

This report gives the results of a survey carried out by the Rhodes-Livingstone Institute in Livingstone, one of a series undertaken in Northern Rhodesia and designed to supply demographic data and to provide some indication of the extent to which Africans are becoming permanently urbanized. After a short preface outlining the aims of the survey, there is a description of Livingstone which is the capital of the Southern Province, the main entry point by rail, the site of the international airport, and the main employment centre for Africans in the southern part of the territory. The Livingstone population which was estimated in the 1951 census to contain 18,000 Africans of whom 9,610 were in employment, is a mixture of tribes speaking many different languages, and with different kinship and political systems and types of economy. During this survey the inhabitants of 10% of the houses, a total of 653 houses with an average of 2·7 persons per house, were interviewed by two Africans of local origin.

The investigation revealed a preponderance of people in the 20–40 age-group and of males over females. The age and sex distribution differed from that in the Copper Belt, probably owing to the presence of a large proportion of men from neighbouring tribes, and to Livingstone being the first town encountered by many immigrants. Investigation of birth-place and tribal origin showed that over 40% of the men and women were from the Barotse Province and 20% from the Southern Province. Only 5% had over 20 years continuous residence in Livingstone, while 68% had 5 years or less. Even among those who had the longest urban residence, very few regarded themselves as permanent town dwellers.

Two-thirds of the adult males were domestic servants or unskilled labourers, nearly 40% were illiterate and only 20% educated beyond elementary school level. Illiteracy is decreasing as shown by the higher standards of education in the younger age-groups. Education and earnings are linked to some extent, for mean wages increased steadily with educational status. Nevertheless, though clerical work attracted the better educated, it was not so well paid as skilled work. Though no accurate estimate was made, the impression was formed that it was difficult for a single man to live on less than £3 or a married couple on less than £5 monthly however low the standard of living. Yet 57% of men were drawing less than £3 and 82% less than £5 10s.

Analysis showed that men were continuously moving from town to town, or from country to town and the reverse. This restlessness was related to

some extent with tribal origin and also with occupation. The white collar workers move more than others, partly owing to official postings. The most skilled and best educated tribal groups changed and moved more frequently. An attempt was made to discover how far people were established in town. Only 1 man in 10 intended to live permanently in town, and a high proportion of the younger men who said so had been brought up in town. The attitude towards permanent town dwelling varied with earnings; irrespective of age, increase in earnings was progressively associated with the tendency to urban life.

Certain broad conclusions are drawn from this survey. The indications are that economic class divisions are already in existence and are stressing rather than bridging previous social class divisions. Indeed, occupation, education and earnings are background factors in a struggle for political leadership, and economic class tends to correspond with tribal group. There are as yet only slight indications of social class becoming a stronger bond than tribal affiliations. The majority of the men intended to spend most of their lives in town but only 10% regarded themselves as permanent dwellers there. The men who were most stabilized in urban areas were also the most mobile between urban areas. This study provides interesting data on a major problem, namely how far the African is developing a civic sense and to what extent he is becoming urbanized. Much more information on a wider range of subjects is available in the report than can be referred to here. *T. H. Davey*

FRAGA DE AZEVEDO, J., GÂNDARA, A. F. & FERREIRA, A. P. Aspectos de Timor em relação com a sua nosologia. [**Aspects of Timor in Relation to the Pattern of Disease**] *Anais Inst. Med. Trop.* Lisbon. 1958, Mar., v. 15, No. 1, 5–34, 29 figs. (26 on 13 pls.) English summary.

Between November 1955 and March 1956, a medical mission from the Institute of Tropical Medicine in Lisbon, under the direction of the senior author, visited Portuguese Timor. The mission had a wide programme which included a survey of prevailing diseases, parasites and mosquitoes and the means of controlling them, nutrition, child development, the incidence of certain cosmopolitan diseases and the collection of specimens for subsequent study and record in the Institute.

During the few months of their stay, the mission carried out a considerable range of studies. These are listed under 20 headings, the first 14 of which are the subject of individual papers in the same issue of the *Anais*: the remainder will appear in subsequent issues.

The present paper is an introduction to the work of the mission, which is to be followed by a permanent mission for the study and control of endemic diseases in Timor. The paper sets out at considerable length details of the geographical, meteorological, biophysical and climatic features of Timor, an account of the population and living conditions and a brief review of prevailing diseases. As these diseases are the subject of separate papers, only the principal findings will be noted here.

Portuguese Timor occupies an area of some 18,900 sq. km. and consists of the eastern half of the island, the territory of Oè-Cusse (which abuts on the

Indonesian part of the island) and the islands of Atauro and Jaco. Much of the territory is mountainous (mostly 400–1,400 metres, with a maximum of some 3,000 metres). It is therefore largely healthy and the most serious endemic diseases are to be found in a small coastal area. The physical features are shown in sketch maps and several photographs which indicate that the country is scenically very attractive.

The population (1950 census) is some 436,000 persons, with a density of 22·96 per sq. km. Most of the people are Malays, there are some Papuans, about 3,000 Chinese and a smaller number of people of European, mixed, Asian and African stocks. The economic standard is low, but the people live in modest comfort as a result of their agricultural pursuits.

As the island is situated in a transitional zone between Australia and Indonesia, it is to be expected that the pattern of disease would be reflected in this factor. In fact, a number of cosmopolitan diseases are found, although some—such as typhoid and paratyphoid fever—are rare. The most important disease is malaria (22,013 cases in 1955, 11,533 in 1957). Tuberculosis, leprosy, venereal disease and dysenteries occur and filariasis and other helminthiases are found. The diseases are discussed fully in the subsequent papers.

It is evident that the full investigation of the diseases of Timor, and their control, will prove a very valuable contribution not only to the welfare of the Territory, but to medical knowledge generally. *H. J. O'D. Burke-Gaffney*

VEEGER, L. M. Papoea-dorpsverzorgsters. Een sociaal-hygiënisch experiment in Nederlands Zuid Nieuw-Guinea. [**Papuan Village Welfare Workers. A Social Hygiene Experiment in Netherlands South New Guinea**] [Thesis.] 143 pp., illustrated. English summary. 1959. Leiden: Laboratorium voor Parasitologie, Rijksuniversiteit, Holland.

This very full monograph was the subject of a doctoral thesis for presentation in the University of Amsterdam. In an introduction and 4 parts it gives an account of an experiment carried out in a sub-district of Merauke in Netherlands New Guinea as part of the effort to improve the socio-hygienic conditions of village populations. It is fully illustrated with photographs having captions in Dutch and English, and contains numerous tables, 5 specimens of registration forms, an outline map and 3 pages of references.

The first part describes a preliminary health and social survey of the people. This showed that development was slow, partly because of tribal tradition, especially among the elders, and partly because of difficult local conditions, which make it hard to approach and influence the people.

As would be expected in an underdeveloped country, maternal and child health were at a low level, and infant mortality was high, despite the absence of many serious diseases which are common in other tropical countries.

The second part sets out at length the means undertaken to relieve the situation. Papuan girls were trained as village welfare workers, with the task of bringing curative and preventive measures to the villagers, especially the mothers and children. The scheme began in 1953, with the training of 4 girls. The programme of the course and the training of the girls are described.

The third part compares this undertaking with similar projects carried out in other countries.

Finally the activities of the village welfare workers are assessed. On the whole, the results were satisfactory and any difficulties which arose can, it is believed, be overcome in the future by careful selection and supervision of new candidates.

It was found essential, in maintaining the enthusiasm and efficiency of the village welfare workers, that regular inspection by district nurses and refresher courses should be carried out [this is a common experience of those who train subordinate staff in the tropics].

The activities of the village welfare workers in the districts concerned have resulted in a considerable improvement in health conditions. Tables showed the reduction of infant mortality and the increase in weight of infants. It is clear that the influence of these workers has resulted not only in better health, but, at least in part, in improved social and economic conditions in several villages.

Costs of the village welfare work are given and, relatively speaking, they are not high. It is pointed out that the village welfare worker now provides permanent, simple care to large groups who formerly could expect little more than an occasional visit by a doctor or male nurse. Such factors encourage extension of this form of village welfare work. *H. J. O'D. Burke-Gaffney*

BOOK REVIEWS

SANDOSHAM, A. A. [L.M.S., Ph.D., F.R.E.S., F.L.S., F.Z.S., F.R.M.S.] **Malariology with special reference to Malaya.** pp. xix + 327, 38 figs. & 19 pls. 1959. London: Oxford University Press, Amen House, Warwick Square, E.C.4. [For the University of Malaya Press, Singapore.] [35s.]

In bringing together the knowledge of malaria and its transmission in Malaya, the author had in mind the needs of the undergraduate medical student and the postgraduate student for the Diploma in Public Health in the University of Malaya. He has therefore sought to make his readers sure of the foundations of the subject.

Starting with a historical and geographical story of the disease in Malaya, he proceeds to very full accounts of the plasmodium and the anopheline, which are notable for their fundamental structure going back to the systems of zoological nomenclature, and for their elaboration of detail. The section on parasitology gives a very full account of each parasite, illustrated by beautiful plates by FIELD, SHUTE and YAP LOY FONG, and proceeding to the epidemiological features of the disease in Malaya. The section devoted to the natural history of mosquitoes is even more detailed. In it the author gives general accounts of the distribution, morphology, life history, physiology and biology of anophelines, which lead up to full statements of each Malayan species,

vector and non-vector, covering the morphology of the adult and larva, prevalence and distribution, bionomics and where appropriate the relation to malaria and methods of control applicable to the species. The foundation which the student is to work on is therefore unquestionably sound.

The second half of the book deals with the application of this knowledge in malaria surveys and control and prevention. These sections bring together the accepted forms of technique for Malaya, dealing with the problems of survey and prevention mainly as local matters to be dealt with in circumscribed conditions, as indeed the intended reader will usually encounter them. They cover such processes as map-making, the taking of spleen and parasite rates and other malariometric indices, the collection of adult and larval mosquitoes, and the various mechanisms of control. The emphasis is strongly placed on traditional techniques; surface, subsurface and tidal drainage, oil as a larvicide, and biological control. Secondary to these come protection by drugs, and with lesser emphasis the use of residual insecticides, which are handled in one and a half pages.

The book closes with very useful appendices covering microscopic, haematological and entomological technique, a summarized aid to diagnosis of parasites, illustrated keys to the adults and larvae of Malayan anophelines, a good bibliography of Malayan standard works, and a glossary.

The readers to whom the author addressed himself have undoubtedly been treated generously and well. They should gain a very sound knowledge of the subject, orientated at first to their own country but sufficiently basic to make elaboration in relation to other countries easy. They would learn the techniques of prevention in the forms most useful to them, for they would perhaps normally be called on to advise on individual and local problems, and this knowledge would give a sure foundation on which the superstructure of eradication and control schemes elsewhere could easily be built. Professor Sandosham has made a notable contribution to the literature of malaria in Malaya.

G. Macdonald

SHAFEI, A. Z. [D.T.M. & H., Ph.D., M.R.C.P.] **Common Endemic Diseases in Egypt.** pp. xii + 170, illustrated. 1958. Cairo: The Renaissance Bookshop Publishers, Egypt.

This is an unpretentious book which reflects the author's views and has been developed from lecture notes to students of the University of Alexandria. As such it has a limited scope and obviously does not seek to complete with the more ambitious textbooks on tropical medicine.

The handling of the subjects is somewhat uneven, but the author becomes more expansive on malaria, amoebiasis and schistosomiasis. In each instance a description of the aetiology is omitted and the life history of the causative parasites hardly touched upon. In his descriptions of treatment the author follows generally accepted lines and in the case of antimalarial synthetic drugs the recommendations of the Colonial Medical Research Committee [see this *Bulletin*, 1954, v. 51, 861], but the bold statement that paludrine is a radical cure for subtertian malaria should not receive universal support, and in blackwater fever no mention is made of the remarkable action of cortisone. In connexion with the action of emetine in the treatment of amoebic dysentery

he places great stress on the cardiac toxicity of this valuable drug and tends to underemphasize its curative properties.

In a comparative analysis of the relative merits of numerous anti-amoebic drugs, he cites the relapse rates of emetine and emetine bismuth iodide as 50% while that of Milibis with chloroquine is given as 4·8% in a dosage of 6 tablets daily for 7–15 days.

On schistosomiasis he becomes most expansive, probably because of the importance it bears on the nosology of Egypt; to this he has devoted 53 pages—one-third of the whole volume. Here the pulmonary, cardiac and vascular changes are probed in great detail.

There is an adequate bibliography and index. *Philip Manson-Bahr*

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Bl.	,,	Blackwater Fever.
B.R.	,,	Book Review.
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Bietti, G. B., (224) (Oph.)
Bigotto, M. R., 664* (Parasit.)
Bigwood, E. J., 871* (Def. Dis.)
Bilek, M., 26 (Typh.)
Bilimoria, D. P., 329* (Hel.)
Bimmer, E., 358 (Tox.)
Binford, C. H., 1247 (Lep.)
Binford, D. H., 537 (Lep.)
Bingle, J. P. 353 (Haem.)
Binson, G., 419* (Mal.)
Biraud, Y., 255 (Reports, etc.)
Birch, C. L., 458 (Hel.)
Bîrzu, N., 939* (Rab.)
Bishop, A., 255 (Reports, etc.)
Biswas, S., 1028 (Chl.)
Bjarnason, O., 1138* (Lep.)
Björk, W., (1184) (Vms.)
Black, R. H., 918 (Mal.)
Blagojevic, M., 1277 (Oph.)
Blaine, G., 225 (Ulc.)
Blake, W. P., (223) (Der.)
Blanc, M., 168 (Lep.), 961* (Hel.)
Blanton, F. S., (1202)* (Ent.)
Blay, E. R., 50 (Am.)
Blenska, W. M., 733* (Lep.)
Blewett, J., 838 (Ys.)
Bliakher, S. L., 721 (Pl.)
Blinov, P. N., 709 (Typh.)
Blum, M. S., (373) (Ent.)
Boccacci, M., (576)* (Ent.)
Bodenstein, O. F., 671* (Ent.)
Boffa, G., (972)* (Haem.)
Boffa, M. C., (972)* (Haem.)
Bogliolo, L., 66 (Hel.)
Boiron, H., 31 (Y.F.)
Bojanowska, A., 143 (Typh.)
Bol. Oficina Sanitaria Panamericana, 629 (Lep.)
Bolin, V. S., 529 (Rab.)
Bolkhovitinova, L. M., 764* (Tox.)
Boman, H. G., (1184)* (Vms.)
Bonetti, F., 545*, 846 (Hel.)
Bonhoure, G., 595*, 806* (Mal.)
Bonjer, F. H., 253 (Reports, etc.)
Bonnet, D. D., 342 (Hel.)
Bonsel, J., 182 (Hel.)
Bonvini, E., 834* (Am.)
Bool, P. H., (1020) (Typh.), 1188* (Tox.)
Boquet, P., 653, 1273* (Vms.)
Bordjochki, A., 767*, 768* (Tox.)
Borgomano, M., (972)* (Haem.)
Borsos, T., 1225* (Tryp.)
Bortolini, G., (953) (Hel.)
Bose, P. N., 296* (Pl.)
Botero, D., 549 (Hel.)
Botha, H. P., 1155* (Hel.), 1219* (Mal.)
Bouisset, L., 16 (Mal.), 282 (Tryp.)
Bourgain, M., (669) (Ent.)
Bourne, T. K. R., 488* (Ent.)
Bower, B. D., 260 (Reports, etc.)
Bowman, M. C., 1286* (Ent.)
Box, E. D., 695 (Mal.)
Boyd, J., 928, 930 (Leish.)
Boyd, J. L., 136*, 927* (Tryp.)
Boyd, J. S. K., 191 (Hel.), 605 (Y.F.)
Bozeman, F. M., 708* (Typh.)
Bradbury, F. R., 1203 (Ent.)
Brading, I., 82 (Haem.)
Branche, G. C. Jr., 1056* (Haem.)
Brand, P. W., 1240 (Lep.)
Brandão, H., (118)* (Mal.)
Bransby-Williams, W. R., 889 (Ent.)
Brant, T. C., 22 (Tryp.)
Bray, R. S., 255 (Reports, etc.), 686 (Mal.)
Breña B., G., 949 (Lep.)
Brener, Z., 286, 600* (Leish.), 322 *bis*, 636, 1156* *bis*, 1250* (Hel.), 1117 (Tryp.)
Brenes, R. R., 170* (Hel.)
Bres, P., 364 (Misc. Dis.), 713* (Y.F.)
Bresani Silva, F., 623 (Lep.)
Bressani, R., (758)* *bis*, 967, 967* (Def. Dis.)
Brezina, R., 292, 604 (Typh.)
Brink, C. J. H,. 14, 1219* (Mal.), 1155 (Hel.)
Brise, H., 872* (Sp.)
Brissaud, H. E., 1063 (Misc. Dis.)
British Red Cross Society, 996 (B.R.)
Brock, D., 1121* (Typh.)
Brooke, J. P., 107 (Ent.)
Brooke, M. M., 45 (Am.)
Brooks, E. A. V., 363 (Misc. Dis.)
Brown, A. W. A., 247 (Ent.), 923 (Mal.)
Brown, E. S., 491* (Ent.)
Brown, H. W., 465, 748* (Hel.)
Brown, J. A. K., 451, 729, 733, 839, 1037 (Lep.)
Brown, K. N., (281)* (Tryp.)
Browne, S. G., 540, 1142 (Lep.)
Bruce-Chwatt, L. J., 120, 688, 1216 (Mal.), 256 (Reports etc.), 1207 (Lab.)
Bruhl, H. H., (563) (Tox.)
Brumpt, E., 1210 (B.R.)
Brumpt, L., 86,* 215 (Haem.), 1210 (B.R.)
Brumpt, V., 215* (Haem.)
Brun, R., 728 (Lep.)
Bruni, F., (857)* (Hel.)
Bruni Celli, B., 1260* (Hel.)
Brygoo, E. R., 307, 337, 1153 (Hel.), 435 *bis*, 1128* (Pl.), 1191 (Der.), (1202) *bis*, (1202)* (Ent.)
Buck, A. A., 183, 746 *bis*, 852*, 853*, 1254 (Hel.)
Buckley, J. J. C., 191, 1171* (Hel.), 250 (Reports, etc.)
Buckner, A. J., 896* (Ent.)
Budden, F. H., 553 (Hel.)
Budtz-Olsen, O. E., 477 (Haem.)
Bueding, E., 848* (Hel.)
Buentello, E., 880 (Tox.)
Buisson, R., 1104* (Mal.)
Bundeali, A., 1027 (Pl.)
Bunjé, H., 1280 (Misc. Dis.)
Buonomini, G., 122 (Mal.), 301, 440 *bis*, 724 (Am.)
Burcham, E., 987 (Ent.)
Burden, G. S., 1290* (Ent.)
Bürgdorfer, W., 822, 936, 937* (Den.)
Burgess, R. W., 122*, 690*, 691 1007* (Mal.)
Burke, J. C., 853* (Hel.)
Burnett, H. S., 544* (Hel.)
Burrell, C. D., 818* (Typh.)
Bursell, E., (1224) (Tryp.)
Burtt, B. D., (112) (B.R.)
Bush, O. B., Jr., 621 (Lep.)
Buso, R., 1055* (Haem.)
Bustamante, M. E., (31) (Y.F.)
Busvine, J. R., 256 (Reports, etc.), 894, 990 (Ent.)
Butt, A. J., (183) (Hel.)
Büttiker, W., (488), (776) (Ent.), 510, 591 (Mal.)
Buttner, A., 64, 462* (Hel.)

* *joint author*

C

Cabanel, G., (302) (Am.)
Cabannes, R. J., 254 (Reports, etc.)
Cabrera, H. A., 159 *bis* (Am.)
Cahill, F. P., 725* (Am.)
Calado, O. B., 68* (Hel.)
Callot, J., 1211 (B.R.)
Camain, R., 31* (Y.F.), 228*, 229* (Misc. Dis.), 965* (Def. Dis.), 980* (Der.)
Cambieri, F., 102* (Parasit.)
Cameron, T. W. M., 854* (Hel.)
Caminopetros, J., 86 (Haem.)
Campbell, J. M., 708* (Typh.)
Campillo, S., C., (31) (Y.F.)
Canalis, A., 408, 409 (Mal.), 719 (Pl.)
Cancio, M., 315* (Sp.)
Canham, P. A. S., 350*, 867* (Def. Dis.)
Cannefax, G. R., 728 (Lep.)
Cannon, D. S. H., 4 (Mal.)
Canova, R., 600 (Leish.)
Cantore, G. P., 479, (562) *ter*, (976) (Vms.)
Cantrell, W., (520), 701 (Tryp.)
Cap, J., 541 (Lep.)
Capocaccia, L., (1193)* (Ulc.)
Capone Braga, P., 367, (573) (Ent.)
Capron, A., (1121)* (Typh.)
Carcassi, U., 774 (Misc. Dis.)
Cardelle, G., 480, 1060 (Tox.)
Cárdenas Vásquez, B., 356 (Tox.)
Carli, G., 569, 1197 (Parasit.)
Carneiro, E., (178)*, 180* (Hel.)
Carneiro, J., Jr., 180* (Hel.)
Caroe, E., 204* (Def. Dis.)
Carr, W. R., (202) (Def. Dis.)
Carrescia, P. M., 129 (Mal.)
Carski, T. R., 1125* (Rab.)
Carson, P. E., (10)*, (1216)* (Mal.)
Carteaud, J. P., 526* (Y.F.)
Carvalho, A., 849* (Hel.)
Carvalho, P. J., (1117)* (Tryp.)
Casals, J., 606* (Y.F.)
Casarosa, A., 569* (Parasit.)
Casey, M., 1121* (Typh.)
Casley-Smith, J. R., 81* (Def. Dis.)
Cassard, H., 977* (Tox.)
Cassel, R., 566* (Misc, Dis.)
Castillo, F., (1178) (Def. Dis.)
Castillo Lindley, E., 1161 (Hel.)
Castillo Plaza, A., (473) (Def. Dis.)
Castrejón, O., 551* (Hel.)
Castrucci, G., 36* *bis* (Rab.)
Čatar, G., 1186 (Tox.)
Cathcart, R., 87* (Haem.)
Catindig, C. T., (433) (Rab.)
Catzel, P., 348 (Def. Dis.)
Causey, O. R., 1228 (Y.F.)
Cavalli, V. (857)* (Hel.)
Cavier, R., (847) (Hel.)
Cawdrey, M. J. H., (423)* (Tryp.)
Cayla, J., 1218 (Mal.)
Ceccaldi, J., 150* *bis* (Rab.), 219* (Tox.)
Cerf, J., 859 (Hel.)
Cerisola, J. A., (424)* (Tryp.)
Černá, Ž., (697) (Mal.)
Cernich, I. R., (353) (Haem.)
Cervone, L., (106) (Ent.)
Chabannes, J., 1034* (Lep.)
Chabaud, A. G., 335 (Hel.)
Chaia, G., 322 (Hel.)
Chakraborty, J., 703* (Leish.)
Chakraborty, M. L., 1271 (Def. Dis.)
Chakravarty, A., 202 (Def. Dis.)
Chakravarty, P. K., 944* (Chl.)
Chambon, L., 627 (Lep.)
Chandler, A. C., 236 (Parasit.)
Chandler, A. H. 563* (Tox.)
Chang, H. L., 565* (Oph.)
Chang, H. T., 70 (Hel.)
Chang, Y. T., 950, 1148 (Lep.)
Chapman, G. B., 734 (Lep.)
Chapman, H., 342* (Hel.)
Chappell, A. C., (637) (Hel.)
Chardome, M., 697* (Mal.), 700* (Tryp.)
Charles, L. J., 1106 (Mal.)
Charles, P., 19* (Tryp.)
Charmot, G., 214, 969* (Haem.), 647, 1176* (Def. Dis.), 957, 1163 (Hel.)
Charnier, M., 58* (Hel.)
Charters, A. D., 1070 (Misc. Pap.)
Chastang, R., 1008 (Mal.)
Chatterjea, J. B., 84, 1181 (Haem.), 1209 (B.R.)
Chatterjee, A. C., 1271* (Def. Dis.)
Chatterjee, A. N., 140 (Leish.)
Chatterjee, B. R., 944 (Chl.)
Chatterjee, H. N., 944 (Chl.)
Chatterjee, K. R., 537, 844 (Lep.)
Chatterjee, S. N., 628 (Lep.)
Chatterji, A., 24 (Leish.)
Chaudhuri, R. N., 726 (Am.)
Chauhan, B. S., (1063) (Parasit.)
Chaussinand, R., 1242 (Lep.)
Chavalier, J., 1220* (Mal.)
Chavanne, P., 731* (Lep.)
Chávez, A., 352 (Haem.)
Chechel'nitskaia, S. E., 817* (Typh.)
Cheema, L. S., 248* (Ent.)
Chee Sinn Lim, 1290* (Ent.)
Cheever, A. W., 546 (Hel.)
Chelărescu, M., 410* (Mal.)
Chen, C. S., 801* (Mal.)
Ch'en, K. H., (199) (Hel.)
Ch'en, K. K., 1045* (Hel.)
Ch'en, W. H., 339* (Hel.)
Chen, W. T., (228)* (Misc. Dis.)
Chen, W. Z., (1159)* (Hel.)
Cheneveau, R., 454 (Lep.)
Cheng. C. L., (463) (Hel.)
Cheong Chee Hock, (1164) (Hel.)
Chernin, E., (1041) (Hel.)
Chernoff, A. I., 651,* (1182) (Haem.)
Chernov, S. G., 719* (Pl.)
Cherry, J. K. T., 1046 (Hel.)
Cherry, L. M., 895 (Ent.)
Chesterman, C. C., 930 (Leish.)
Chevrier, L., 715* (Rab.)
Chhuttani, P. N., 1161 (Hel.)
Chi, L. W., 1041* (Hel.)
Chiang, P. J., 327* (Hel.)
Chiang, S. C., 181* (Hel.)
Chiari, E., 322* (Hel.)
Chibrikova, E. V., 1233 (Chl.)
Ch'ien, H. C., 665 (Parasit.)
Chiles, W. D., 483 (Heat Str.)
Ch'in, Y. T., 1200 (Ent.)
Chinese Med. J., (69), 864 (Hel.)
Chitre, R. G., (559) (Def. Dis.)
Chlouverakis, C., 86* (Haem.)
Chopra, I. C., 1209 (B.R.)
Chopra, R. N., 1209* (B.R.)
Choremis, C., 876, 1272 (Haem.)
Chou, S. N., 1158* (Hel.)
Chou, T. Y., 1165* (Hel.)
Christian, S. H., 269* (Mal.)
Christie, M., 12, 270, 385 (Mal.)
Christol, D., 1063 (Misc. Dis.)
Chu, C. C., 636 (Hel.)
Chu, F. I., 245 (Ent.)
Chu, S. H., 186 (Hel.)
Chugh, K. S., 1161* (Hel.)
Cicardo, V. H., (479) (Vms.)
Ciplea, A., 425* (Leish.)
Ciucă, A. M., 256 (Reports, etc.)
Ciucă, M., 410, 922 (Mal.)
Clark, P. H., 26*, (289)* (Typh.)
Clarke, G. H. V., 903 (B.R.)
Clarke, T. H., 1205 (Ent.)
Clary, J., 1146* (Lep.)
Clastrier, J., (490)*, (669), (1068), (1202) (Ent.)
Clearkin, P. A., 209 (Haem.)
Cleve, E. A., (359) (Der.)
Close, H. P., 87* (Haem.)
Close, J., 348 (Def. Dis.)
Clyde, D. F., 272, (691), 692 (Mal.)
Coatney, G. R., 122 (Mal.)
Cochran, D. G., 1205* (Ent.)
Cochrane, R. G., 496 (B.R.), 625 (Lep.)
Coda, D., 1156* (Hel.)
Coelho, M. de V., 177, 177*, 1157* (Hel.)
Cohen, F., 971*, 1181 (Haem.)
Cohen, G., 330 (Hel.)
Cohn, Z. A., 708 (Typh.)
Coker, W. Z., 888 (Ent.)
Colas-Belcour, J., (991) (Ent.)
Colbourne, M. J., 919 (Mal.)
Cole, M. M., 26, (289) (Typh.), 670, 1290 (Ent.)
Cole, W. R., (871)* (Def. Dis.)
Coleman, N., 355*, 480* (Tox.)
Coles, R. M., 899 (Misc. Pap.)
Colhoun, E. H., 672 (Ent.)
Colless, D. H., 242, (669), (1285) (Ent.), 337* (Hel.)
Collier, L. H., 95, 772 (Oph.)
Collier, W. A., 525*, 605, 714, 1122, 1228, 1229 (Y.F.)

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Colling, M., 227 (Misc. Dis.)
Collins, R. F., 68, 181* (Hel.)
Collomb, H., 254 (Reports etc.,), 704*, 705* (Leish.)
Colonial Office, 677 (Reports, etc.), 885 (Ent.)
Colson, J. A., 833 (Am.)
Coluzzi, A., 122* (Mal.)
Coluzzi, M., (117), 124 (Mal.)
Colwell, J. A., 981 (Der.)
Combiesco, D., 287 (Typh.)
Combrink, H. J., 1155* (Hel.), 1219* (Mal.)
Combs, G. F., (1176)* (Def. Dis.)
Comité de L'Entente Française, 1172 (Hel.)
Commission for Technical Co-Operation in Africa South of the Sahara, 18 (Tryp.)
Congo Belge, 375 (Reports, etc.)
Conley, C. L., 651* (Haem.)
Conradie, F. M., 649* (Haem.)
Consigli, R. A., 27* (Typh.)
Constantinescu, G. 410* (Mal.)
Constantinescu, N., 939 (Rab.)
Constantinescu, P., 410* (Mal.)
Conte, V. P., 1252* (Hel.)
Contreras, F., 252 (Reports, etc.), 446, (843) (Lep.)
Contreras, F., Jr., 841, (843)*, 1144* (Lep.)
Convit, J., 931 (Leish.)
Cook, J., (1246) (Lep.)
Cook, M. K., 92, 93, 219, 769 (Tox.)
Cooke, W. T., 475 (Sp.)
Cook-Sup So, 1031 (Am.)
Cooney, M. K., 220, 220* (Tox.)
Cooper, T. V., 644* (Hel.)
Cooper, W., 694* (Mal.)
Coquelet, M. L., 86*, 215* (Haem.)
Coraggio, F., 824* (Rab.)
Corbet, P. S., (370), 1285* (Ent.)
Corbo, S., 234*, 1064* (Parasit.)
Coritsoglou, A. C., 750 (Hel.)
Corkill, N. L., 654 (Vms.)
Cosnand, G., 495 (Reports, etc.), (1276) (Oph.)
Cornet, L., 614 (Am.)
Corrêa, M. O. A., 751*, (Hel.) 931 (Leish.)
Correa, P., 1192 (Der.)
Correa, R. R., (1117) (Tryp.), 1156 (Hel.)
Corrêa Fleury, G., 751* (Hel.)
Corticelli, B., 429*, 1227* (Typh.)
Costa, A. 816* (Typh.)
Costa, D., 757 (Def. Dis.)
Costa, L., (339)* (Hel.)
Costello, M. J., 658 (Der.)
Costin, P., (269)* (Mal.)
Cotaesco, E., 295 (Der.)
Cotter, H., 476, 1181* (Haem.)
Coudert, J., 854 (Hel.)
Courbil, J., 1193* (Ulc.)
Courshee, R. J., (1291) (Ent.)
Courson, B., 724* (Am.)
Coutinho, J. O., 835 (Am.)
Coutinho-Abath, E., 69, 1157* (Hel.)
Cowper, S. G., 595 (Mal.)
Cox, H. W., 806 (Mal.)
Coyne, F. P., 256 (Reports, etc.)
Cradock-Watson, J. E., (1272) (Haem.)
Cravioto, J., 347, 647* (Def. Dis.)
Crockett, E. J., 1182* (Haem.)
Crosskey, M. E., 1266* (Hel.)
Crosskey, R. W., 78, 555, 1266 (Hel.)
Crotty, J. M., 760 (Haem.)
Crowden, G. P., 253 (Reports, etc.)
Cruz Ferreira, F. S., 252 (Reports, etc.)
Cruz Krohn, J., 352* *bis* (Haem.)
Cuboni, E., 1274 (Tox.)
Cuevas, C., (1178)* (Def. Dis.)
Curbelo, A., 480* *bis*, 1060* *bis* (Tox.)
Cvjetanovic, B., 251 (Reports, etc.)

D

D'Abrera, V. St. E., 485, 982, (983), 1281 (Misc. Dis.)
Da Cunha, J. F., 255 (Reports, etc.)
Da Cunha Pinto, M. R., 257 (Reports, etc.)
Dadiotou, D. D., 1248* (Hel.)
Da Fonseca, P., 1116* (Tryp.)
Dagert, B. C., 462* (Hel.)
Daggy, R. H., 256 (Reports, etc.), 1213 (Mal.)
Da Graca, M. M., 1116* (Tryp.)
Dahnovici, V., 1007* (Mal.)
Daikos, G. K., 1029* (Am.)
D'Alesandro, P. A., (1116) (Tryp.)
D'Alessandro, G., 515 (Mal.)
D'Ambrosio, R., (857) (Hel.)
Damiean-Gillet, M., 344 (Def. Dis.)
Danaraj, T. J., 337 (Hel.), 983, 1061 (Misc. Dis.)
Danauskas, J. X., 814* (Typh.)
Daniels, R. A., 70* (Hel.)
Dannenberg, A. M., Jr., (365) (Misc. Dis.)
Darbon, A., (637) (Hel.)
Da Rocha Passos, M. C., Jr., 223 (Der.)
Das, N., 443 (Am.)
Das Gupta, A. K., 970* (Haem.)
Da Silva, J. F., 1156* (Hel.)
Da Silva, J. M., (924), (1116) (Tryp.)
Da Silva, J. R., 254 (Reports, etc.)
Da Silva, L. S., 1061* (Misc. Dis.)
Daswani, H. D., 874* (Haem.)
Datta, S. P., 377* (Reports, etc.), 594* (Mal.)
D'Aubenton, F., 961 (Hel.)
Dauer, C., 1226* (Leish.)
Dave, P. J., 309 (Hel.)
Davey, D. G., 20*, 518 (Tryp.)
Davey, T. F., 450, 537, 542, 1146, 1245 (Lep.)
Davey, T. H., 226 (Misc. Dis.)
David, H. V., 340* (Hel.)
David-Chaussé, J., (954)* (Hel.)
Davidson, G., 273, 419 (Mal.), 1203* (Ent.)
Davies, A. M., 1124 (Rab.)
Davies, J. B., 133 (Tryp.)
Davies, J. N. P., 722 (Chl.), 899*, 1207 (Misc. Pap.)
Davies, M., 670 (Ent.)
Davis, D. E., (344) (Hel.), 717 (Rab.)
Davis, D. H. S., (780) (Ent.)
Davis, M., 68*, 181* (Hel.)
Davison, A. R., 730, 844*, 1145 (Lep.)
Day, A. J., 81* (Def, Dis.)
De, S. N., 44, 1028 (Chl.)
De Almeida, A. M., 65* (Hel.)
De Almeida, G. R., 68* (Hel.)
De Almeida, J. O., 1037 (Lep.)
De Almeida Franco, L. T., 561* (Haem.)
De Almeida Neto, E., 453 (Lep.)
Dean, N. M. B., 493 (Misc. Pap.)
Dean, R. F. A., 110* (Misc. Pap.), 871*, 1177 (Def. Dis.)
De Andrade, L. C., 1143 (Lep.)
De Andrade, R. M., 118 *ter* (Mal.), (321) (Hel.)
De Andrade Mendonça, I., 137* (Tryp.)
De Andrade Silva, M. A., 1112 (Tryp.)
Deane, L. M., 23 (Leish.), 251 (Reports, etc.), 425 (Tryp.)
Deane, M. P., 23* (Leish.), 425* (Tryp.)
De Blasi, R., (534) (Am.)
Debré, R., 1063 (Misc. Dis.)
Debry, G. 145* (Typh.)
De Bustamante, F. M., 9, (1103) (Mal.), 20, 22*, 139 (Tryp.)
De Carneri, I., 323 (Hel.), 441, 534, 535, 536, 617, 832 (Am.)
De Carvalho, A. G., 21, 139* (Tryp.)
De Carvalho, A. M., 1114* (Tryp.), 1152 (Hel.)
Dedmon, R. E., 1183 (Haem.)
De Dominicis, A., 235* (Parasit.)
Dedusenko, A. I., 1119* (Typh.)
Deering, D., 513* (Mal.)
DeFeo, C. P., Jr., 658* (Der.)
De Freitas, C. A., 94 (Oph.), 297 (Pl.)
De Freitas, U., 1143* (Lep.)
De González, A. A., 445 (Lep.)
De Groot, A. P., (579)* (Misc. Pap.)
De Hollanda, H., 254 (Reports, etc.)

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De Hollanda, H. H., 757* (Def. Dis.)
Deichmann, W. B., 89*, 90 (Vms.)
De Jager, H., 1194* (Misc. Dis.)
De Jongh, R. T., 1115* (Tryp.)
Delannay, M., 875 (Haem.)
Delanoe, G., 474, (1270)* (Def. Dis.)
De La Peña, C., 647* (Def. Dis.)
Delas, A., 413* (Mal.)
Delbrouck, J., 210 (Haem.)
Deliwala, C. V., 1027* (Pl.)
Dellien, H., 1243* (Lep.)
Del Pianto, E., 537 (Lep.)
Del Razo, M., (860)* (Hel.)
De Lucena, D. T., 21, (282), 424 (Tryp.)
Del Vecchio, P., 1239 (R.F.)
Delwiche, E. A., 824 (Pl.)
Demaeyer, E. M., 347, 349, 557 (Def. Dis.)
Demarchi, J., 969* (Haem.)
Demarchi, M., 208 (Haem.)
De Maria, B., 72 (Hel.)
De Medeiros, L. do C. M., 952* (Hel.)
De Meillon, B., 314, 461 (Hel.), 516* (Mal.)
De Meira, M. T. V., 1204, (1204) (Ent.)
DeMello, E. R., 617 (Am.)
Demina, N. A., 277 (Mal.)
De Montaigne, E. L., 621 (Lep.)
De Moraes, J. G., 180* (Hel.)
De Morais, T., 1065 *ter* (Parasit.), 1152 *bis* (Hel.)
Denisenko, L. K., 829* (Chl.)
Denisoff, N., 464 (Hel.)
Deodhar, A. D., 646 (Def. Dis.)
De Oliveira, N. M., 756 (Def. Dis.)
De Oliveira Musacchio, M., 137* (Tryp.)
De Paola, D., 24*, 140*, (427)* (Leish.)
De Paula, O. R., (1206)* (Ent.)
Depieds, R., 704, 705* (Leish.), (972), (972)* (Haem.)
Depoux, R., 150, 150*, (Rab.) 219*, 977* (Tox.), 1176* (Def. Dis.)
Deran, C., (1121)* (Typh.)
Derbes, V., 204* (Def. Dis.)
Derbes, V. J., 1273* (Vms.)
Dereani, C., 1276 (Oph.)
De Rodaniche, E., 1122 (Y.F.)
De Roever-Bonnet, H., 148, 525*, 605*, 714*, 1122*, 1228*, 1229* (Y.F.), 221 (Tox.)
De Romaña, M. S., 138* (Tryp.)
De Rook, H., 1167 (Hel.)
Deroover, J., 132 (Tryp.)
Derrick, E. H., (935), 936*, 1019 (Typh.)
Derrien, Y., (972)* (Haem.)
Descous, S., (1202)* (Ent.)
Deslandes, N., 1250* (Hel.)
Desmonts, G., 978 (Tox.)
De Sousa, H. T., 1204*, (1204)* (Ent.)
De Souza-Araujo, H. C., 840, 1039, 1146 (Lep.)
De Souza Lima, L., 629 (Lep.)
Destombes, P., 252 (Reports, etc.), 656, 980 (Der.)
Detrait, J., 653* (Vms.)
De Traverse, P. M., 86, 215* (Haem.)
Devadatta, S., 1282* (Misc. Dis.)
Devakul, K., 103* (Parasit.), 276 (Mal.)
Devers, R., (1207) (Ent.)
Devi, K. P., 873* (Haem.)
Devignat, R., 582 (B.R.)
De Vries, A., 771* (Vms.)
DeWitt, W. B., 635* (Hel.)
Dhage, K. R., 309* (Hel.)
Dhatt, M. S., 248* (Ent.)
Dhupar, H. L. 163* (Am.)
Diamond, L. K., 86*, 971* (Haem.)
Diamond, L. S., 283, 702* (Tryp.)
Dias, E., 599 (Tryp.)
Dias, J. A. T. S., (990), 1288 (Ent.)
Dias, N. P., 599* (Tryp.)
Diaz Carral, M. del C., 480* 1060* (Tox.)
Diaz Marin, J., 409, (1213) (Mal.), 439 (Am.)
Díaz Nájera, A., 109* (Ent.)
Diaz Vazquez, A., 1012 (Tryp.)
Dibble, E. H., 1232* (Rab.)
Di Bello, R., (465) (Hel.)
Dickson, W., 642 (Hel.)
Di Egidio, M., 631 (Hel.)
Diggs, L. W., 254 (Reports, etc.)
Dimulescu, C., 754* (Hel.)
Dinculescu, M., 420* (Mal.), (935)*, 1227* (Typh.)
Diniz, O., 629, 1038 (Lep.)
Di Perri, T., (273) (Mal.)
DiPrisco, E., 1162,* (1162)* (Hel.)
Di Salvo, A. F., 146 (Typh.)
Dishon, T., 654* (Vms.)
Dissanaike, A. S., 329 (Hel.)
Dixit, N. S., (410) (Mal.)
Dodin, A., (1128) (Pl.), (1202), (1202)* *bis* (Ent.)
Dodu, S. R. A., 560 (Haem.)
Doery, H. M., 354 (Vms.)
Dolgov, N. V., 430, 817 (Typh.)
Dolkart, R. E., 723 (Am.)
Domaradskii, I. V., 829 (Chl.)
Domato, R. M., 283* (Tryp.)
Domergue, C. A., (1183) *bis* (Vms.)
Domiczstyczyńska, B., 143* (Typh.)
Dominici, L. M., 664 (Parasit.)
Donckaster, R., 162*, 1136 (Am.), 330, 855* (Hel.)
Donnelly, J., (490)* (Ent.)
Donoso, F., 855 (Hel.), 1136* (Am.)
Doraiswamy, T. R., 345* (Def. Dis.)
Dorantes, S., 551* (Hel.)
Dormer, B. A., 251 (Reports, etc.)
Douchet, C., (641)* (Hel.)
Doull, J. A., 629, 844, 1242 (Lep.)
Dowling, M. A. C., 1109 (Mal.)
Downs, C. M., 27*, 709* (Typh.)
Downs, W. G., 252 (Reports, etc.)
Draganesco, N., 1231* (Rab.)
Drăgănescu, N., 941* (Rab.)
Draper, C. C., 917* (Mal.)
Dressler, H. R., 707* (Typh.)
Drewett, S. E., 450* (Lep.)
Dreyfus, B., (85)* (Haem.)
Dreyfus, J. C., 215*, (874) (Haem.)
Dreyfuss, F., 352 (Haem.)
Driver, A. F. M., 483 (Heat Str.)
Drouhet, E., 252 (Reports, etc.)
Dube, B., 875 (Haem.)
Dubinskaia, G. I., 576 (Ent.)
Dubois, A., 198* (Hel.), 379, 999 (B.R.), 540 (Lep.)
Dubois, R., 871* (Def. Dis.)
Ducasse, P., 279* (Tryp.)
Dudkova, V. K., 1233* (Chl.)
Duke, B. O. L., 197, 342 (Hel.)
Dumas, N., 142* (Typh.)
Dupin, H., 965* (Def. Dis.)
Duport, M., 593 (Mal.)
Durasova, M. N., 154* (Rab.)
Durrance, J. R., 482* (Der.)
Dusanic, D. G., (1158) (Hel.)
Dutour, J., 656* (Der.)
Duval, L., 524* (Typh.)
Duvernay, C. E., (1270) (Def. Dis.)
Dymowska, Z., (508)* (Mal.), 768* (Tox.)
Dzhaparidze, M. N., 826 (Pl.)

E

Earle, K. V., 486 (Misc. Dis.)
East Africa High Commission, 257, 494, 781, 1294 (Reports, etc.), 588 (Mal.)
East African Trypanosomiasis Research Organization, (112) (B.R.)
Eden, W. G., 371* (Ent.)
Edeson, J. F. B., 121* (Mal.), 191 *bis*, 191*, 551*, 1171 (Hel.), 251 (Reports, etc.)
Edge, G. A., 1229 (Rab.)
Edge, N. D., 68* (Hel.)
Edington, G. M., 1050 (Def. Dis.)
Edmunds, P. N., (304)* (R.F.)
Edozien, J. C., 363 (Misc. Dis.), 759 (Haem.)
Edwards, P. R., Jr., 613*, 831* (Am.)
Edwards, R., 838* (Lep.)
Ehrenworth, L., 70 (Hel.)
Eisler, D. M., 942 (Pl.)
Eklund, C. M., 35, 936*, 937* (Den.)
El Azzawi, J. A. H., 171 (Hel.)
Eldefrawi, M. E., 1204 (Ent.)

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El-Dewi, S., 762 (Haem.)
Eldon, J. H., (1148) (Lep.)
Elek, S. D., 168* (Lep.)
Eliseev, L. N., 287 (Leish.)
El-Kateb, H., 953 (Hel.)
Elliott, R., 418, 804* (Mal.)
Ellis, F. P., 883 *bis* (Heat Str.)
El-Mawla, N. G., 172* (Hel.)
Elmore, C. M., Jr., 489 (Ent.)
El Nagah, A. M., 741* (Hel.)
El-Nagar, H., 62 (Hel.)
El Raggal, M., 954 (Hel.)
El Raii, F., 60* (Hel.)
Elsdon-Dew, R., 160*, 444*, 832*, 1235* (Am.)
El-Shawarby, K., 1072* (Reports etc.)
El-Ziady, S., 837 (R.F.)
Emam, A. M., 486 (Misc. Dis.)
Emanueli, A., 1183* (Haem.)
Embil Puig, J., (355)* (Tox.)
Emmons, P., (663) (Misc. Dis.)
Encyclopédie Médico-Chirurgicale, 1063 (Misc. Dis.)
Epstein, E., 1160 (Hel.)
Erasmus, F. J., 1155* (Hel.)
Erlandson, M. E., 353, 652, (763), (970) (Haem.)
Escallón, A., 1013* (Tryp.)
Espejo, H., 1252* (Hel.)
Espinosa, J., (860)* (Hel.)
Essawy, M. A., 460* (Hel.)
Essawy, M. A. E., 1072* (Reports, etc.)
Esteves de Sousa, A., 1224 (Tryp.)
Esteves de Sousa, A. de A., (924) (Tryp.)
Ethiopia, 948 (Lep.)
Etteldorf, J. N., 1120* (Typh.)
Evans, A. D., 818 (Typh.)
Evans, A. S., 848 (Hel.)
Eydan, R., (671) (Ent.)
Eyles, D. E., 480*, 1007* (Mal.), 1189* (Tox.)
Eyraud, M., 12*, 419* (Mal.), 367* (Ent.)

F

Fabiani, G., 130, 595, 806 (Mal.)
Fahmi, T., 762* (Haem.)
Faillace de Leon, R., 938 (Rab.)
Fair, J. R., 221 (Tox.)
Fairchild, G. B., 669, 777 (Ent.)
Fairclough, R., 423 (Tryp.)
Falcão, P., 703* (Tryp.)
Falcone, R., 1192* (Der.)
Falconer, D. J. B., (615) (Am.)
Fales, J. H., 671 (Ent.)
Fanci, U., 465 (Hel.)
Fang, J. C., 70*, 1158* (Hel.)
Fang, K. Y., 1158* (Hel.)
Faraco, F., (330) (Hel.)
Farhan, I., (1258) (Hel.)
Farid, Z., 741 (Hel.)
Farjo, P. D., (300) (Am.)
Farkas-Himsley, H., (158) (Chl.)
Farooq, M., 325* (Hel.)
Farquhar, J. W., (304) (R.F.)
Farrell, J. J., 90* (Vms.)
Fauran, P., (744)* (Hel.), (896)* (Ent.)
Faure, A., 705* (Leish.)
Faust, E. C., 251 (Reports, etc.), 1236 (Am.)
Favier, G., (671) (Ent.)
Fawdry, A. L., 727 (Lep.)
Fay, R. W., 109, 489* (Ent.)
Fedorova, N. I., (429)*, 430*, 711* *bis*, 819 (Typh.)
Feldman, H. A., (92) (Tox.)
Felsani, F., 1193 (Misc. Dis.)
Felsenfeld, O., 157 (Chl.)
Feng, L. C., 341 (Hel.), 589* (Mal.)
Feng, S. Y., 799* (Mal.)
Fenton, J. C. B., (1272)* (Haem.)
Ferguson, A. D., 972 (Haem.)
Ferguson, F. F., 460, 850* (Hel.)
Fernand, V. S. V., 350* (Def. Dis.)
Fernandez, E., 344 (Hel.)
Fernandez, J. V., 844* (Lep.)
Fernández Baquero, G., (1191) (Der.)
Fernández Nafria, A., 128 (Mal.)
Fernando, N., 189 (Hel.)
Fernando, P. V. D., 642 (Hel.)
Ferrara, A., 1274 (Tox.)
Ferraz, D. M., 1167, 1167* (Hel.)
Ferreira, A. P., 1103* (Mal.), 1152*, 1165* (Hel.), 1176* (Def. Dis.), 1198* (Parasit.), 1240* (Lep.), 1296* (Reports, etc.)
Ferreira, C. S., 1151* (Hel.)
Ferreira, J. M., 178, 1151* (Hel.)
Ferreira, M., (1130) (Chl.)
Ferreira, M. O., 125* (Mal.), (1202)* (Ent.)
Ferreira Neto, J. A., 104*, 105*, (1202)* (Ent.), 125* (Mal.)
Ferro-Luzzi, A., 647, 755 (Def. Dis.)
Ferro-Luzzi, G., 200, 203 (Def. Dis.)
Fichez, 146* (Typh.)
Field, T. E., 650* (Haem.)
Fiese, M. J., 1209 (B.R.)
Figares, E., 161*, 162* (Am.)
Filippova-Nutrikhina, Z. L., 764, 769* (Tox.)
Findlay, J. D., 362 (Heat Str.)
Fiorillo, A. M., 138* (Tryp.)
Fischer, D. A., 482 (Der.)
Fisk, F. W., (109) (Ent.)
Fisken, R. A., 130* (Mal.)
Flatmark, T., 80 (Hel.)
Flemings, M. B., 778 (Ent.)
Flesh, Y., 654* (Vms.)
Floch, H., 55 *bis*, 626, 629, 731 (Lep.), 124 (Mal.), 336, (744) (Hel.), (896) (Ent.)
Flores, W., 1013* (Tryp.)
Flores-Barroeta, F., 1135 (Am.)
Flores Espinosa, J., (860) (Hel.)
Flynn, A. D., 371 (Ent.)
Foley, A. R., 524* (Typh.)
Foll, C. V., 581 (Reports, etc.), 1135 (Am.)
Follis, R. H., Jr., (101) (Misc. Dis.), 473* (Def. Dis.)
Fonds Reine Élisabeth pour L'Assistance Médicale aux Indigènes (FOREAMI), 540 (Lep.)
Fong, H. W., 627* (Lep.)
Food and Agriculture Organization of the United Nations, 200, 965, 1271 (Def. Dis.)
Forattini, O. P., 988 (Ent.), 1226 (Leish.)
Ford, J., 251 (Reports, etc.)
Forney, R. B., 49* (Am.)
Forsyth, D. M., 317, 633 (Hel.)
Foster, F. H., 748 (Hel.)
Foster, R., 809, 811, 924 (Tryp.)
Foucher, G., 1114* (Tryp.)
Fournier, J., 454* (Lep.)
Fourrier, A., 37* (Rab.)
Fowler, M., 1283 (Misc. Dis.)
Fox, I., 242 (Ent.)
Fox, R. H., 362 (Heat Str.)
Fox, R. M., 919 (Mal.)
Foy, H., 638 (Hel.), 1053 (Haem.)
Fraga de Azevedo, J., 257, 1296 (Reports, etc.), 952, 1152, 1165 (Hel.), 1103 (Mal.), 1176 (Def. Dis.), 1198 (Parasit.), 1240 (Lep.)
Fraimow, W., 87 (Haem.)
Franchini, F., 1252* (Hel.)
Franchino, E. M., 427* (Leish.)
Francis, R. S. R., (331)* (Hel.)
Franco, O., 432 (Y.F.)
Frank, G. H., 314* (Hel.)
Franz, K. N., 471* (Hel.)
Fraser, B. N., 1279 (Misc. Dis.)
Fraser, J. M., 254 (Reports, etc.)
Frazer, A. C., (82) (Sp.)
Freed, J. E., 750*, 752* (Hel.)
Freedman, L., 45, 160*, 1235 (Am.)
Freedman, M. L., 719 (Pl.)
Freeman, F., 160* (Am.)
Freeman, P., 930 (Leish.)
Freire, R. S., (139) (Tryp.)
Frenken, J. H., 955 (Hel.)
Frick, L. P., 182* (Hel.), 233* (Parasit.)
Friedheim, E. A. H., 743 (Hel.), 924, 1115, 1222 (Tryp.)
Friedman, S. B., 40* (Pl.)
Froehlich, M., 651* (Haem.)
Frontali, N., (892) (Ent.)
Frye, W. W., 725, 830* (Am.)
Fuhrer, B., 1023*, 1213* (Rab.)
Fuhrmann, G., 559 (Def. Dis.)
Fuki, A. D., 1121* (Typh.)
Fukuda, H., 870* (Def. Dis.)
Fukuda, M., 843, 1036 (Lep.)
Fukui, G. M., 437*, 824* (Pl.)
Fukushima, S., 361*, 659* (Oph.)
Fülöp, T., 736 (Hel.)
Fulton, J. D., 812, 1113 (Tryp.), 930 (Leish.), 1190* (Tox.)

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Furlong, M., 487* (Ent.), 960 (Hel.)
Furman, D. P., 897 (Ent.)
Fürst, C., 549* (Hel.)
Furtado, T. A., 564 (Der.)
Furtunescu, G., (1200) (Ent.)
Furusho, Y., 529* (Rab.)

G

Gabaldón, A., 257 (Reports, etc.), 1217 (Mal.)
Gahan, J. B., 490*, 666*, 893* (Ent.), 803 (Mal.)
Gail, R., 1183 (Vms.)
Gaines, T. B., 720* (Pl.)
Gaitonde, B. B., 967 (Def. Dis.), 1195* (Misc. Dis.)
Galindo, P., 148, 1122 (Y.F.)
Gall, Z., 291* (Typh.)
Galley, R. A. E., 256 (Reports, etc.)
Galliard, H., 1063 *ter* (Misc. Dis.)
Galliera, A., 846* (Hel.)
Galvão, A. B., (1205)* (Ent.)
Game, B. P., 1135* (Am.)
Gamet, A., 631* (Hel.), 1016 (Typh.)
Ganapathy, S. N., (774) (Misc. Dis.)
Gancarz, Z., 326 (Hel.)
Gândara, A. F., 1103* (Mal.), 1152*, 1165* (Hel.), 1176* (Def. Dis.), 1198* (Parasit.), 1240* (Lep.), 1296* (Reports, etc.)
Gaon, J., 291 (Typh.)
Garabedian, G. A., 856 (Hel.)
Garcia, E. G., 325* (Hel.)
Garcia, S., 352* *bis* (Haem.)
García de Onís, F., (1258) (Hel.)
Garcin, D., 1034* (Lep.)
Gardelle, G., 480* (Tox.)
Gardner, A. F., (884) (Misc. Dis.)
Gardner, E. S., 580* (Reports, etc.)
Gardner, E. W., 45* (Chl.)
Gardner, R. F. R., 580 (Reports, etc.)
Gariou, J., 700* (Tryp.)
Garms, R., 515*, 1107 (Mal.)
Garnham, P. C. C., 132, 694 (Mal.), 928, 930 *bis* (Leish.), 992 (Misc. Pap.), 1286* (Ent.)
Gasteau, T., 972* (Haem.)
Gaud, J., 59, 184, 312 (Hel.)
Gavrilă, I., 433 (Rab.)
Gay-Prieto, J., 728* (Lep.)
Gazizova, G. R., 817* (Typh.)
Geber, M., 110 (Misc. Pap.)
Geekie, H. A. W., 160 (Am.)
Geerts, J., 837 (R.F.)
Geigy, R., 166 (R.F.), 1222 (Tryp.)
Gelfand, M., 61*, 1040 (Hel.), 661 (Misc. Dis.), 770 (Der.), 1071, 1208* (Misc. Pap.)
Gel'ttser, R. R., 303 (R.F.)
Gemmell, M. A., (330) (Hel.)
G.E.N. Caracas, 1196 (Parasit.)
Gentzkow, C. J., 248* (Lab.)
Georgescu, M., (885)* (Parasit.)
Gerald, P. S., 86, (216), 971* (Haem.)
Gerasiuk, L. G., 1119* (Typh.)
Gerbec, M., 604, 710* (Typh.)
Gerloff, R. K., 937 (Der.)
Gerner-Smidt, M., 307 (Hel.)
Gerolt, P., 1205 (Ent.)
Gersdorff, W. A., 490 (Ent.)
Ghalioungui, P., 1072 (Reports, etc.)
Ghendon, Y. Z., 1232* (Rab.)
Gherman, I., 425* (Leish.) 1211 (B.R.)
Ghosh, J. J., 140* (Leish.)
Ghosh, P. K., 857 (Hel.)
Ghosh, S., 52*, (305) (Lep.)
Gibson, C. L., 355, 480* (Tox.)
Gibson, F. D., 509, 688* (Mal.)
Gibson, T. E., 854, 1256 (Hel.)
Gigitashvilli, M. S., 1257* (Hel.)
Giglioli, G., 256 (Reports, etc.), 1047 (Def. Dis.)
Gilbert-Desvallons, Y., 146* (Typh.)
Gil Collado, J., (777) (Ent.)
Gilles, H. M., 103* (Parasit.), 256 (Reports, etc.), 1168 (Hel.)
Gillett, J. D., 370*, 1285 (Ent.)
Gillies, M. T., 271 (Mal.), 574, 665 (Ent.)
Gillman, T., 350, 867, 1051, 1052* (Def. Dis.)
Gilroy, A. B., 693, 694 (Mal.)
Gima, I., 410* (Mal.)
Gingrich, W. D., 695* (Mal.)
Ginsburg, H., 654* (Vms.)
Giraldo, L. E., 251* (Reports, etc.)
Girard, G., 156, 827, 1128, 1129 (Pl.)
Girard, G. G., (1171)* (Hel.)
Gird, E., 1023*, 1231* (Rab.)
Giroud, P., 99* (Misc. Dis.), 142 (Typh.), 253 (Reports, etc.), 772, 772*, 773, 1061* (Oph.)
Gispen, R., 142* (Typh.)
Gitter, S., 771*, 974*, 1273* (Vms.)
Glazko, A. J., 130* (Mal.)
Goble, F. C., 136, 927 (Tryp.)
Gökberk, C., 804 (Mal.)
Gokhale, B. B., 448, 620 (Lep.)
Goldberg, E., (80) (Hel.)
Goldstein, R. H., 725 (Am.)
Goldwasser, R. A., 932 (Typh.), 1125 (Rab.)
Golinevich, E. M., (602)*, 708* (Typh.)
Golinevitch, H., 253* (Reports, etc.)
Golovatskaya, G. I., 764* (Tox.)
Goma, L. K. H., 241 (Ent.)
Gomes, F. E. C., 250 (Reports, etc.)
Gómez, F., 647* (Def. Dis.)
Gomez, J. J., 549* (Hel.)
Gómez Leal, A., 352, 352* (Haem.)
Gómez Mendoza, A., 841* (Lep.)
Gómez Orozco, L., 1039* (Hel.)
Gon, F., (650) (Haem.)
Gongora, J., 1174 (Def. Dis.)
Gonon, M., 435, 435* (Pl.)
Gonzales-Mugaburu, L., 1045 (Hel.)
Gonzalez Barranco, D., 645* (Hel.)
González Ochoa, A., 564 (Der.)
Goodwin, L. G., 25* (Leish.), 639 (Hel.)
Gopalan, C., 558, 1178* (Def Dis.)
Gordadze, G. N., 1257 (Hel.)
Gordienko, E. G., 434* (Rab.)
Gordon, R. M., 191 (Hel.), 698 (Tryp.)
Gorman, G. W., 702* (Tryp.)
Gorshunova, L. P., 153 (Rab.)
Goszczyńska, K., 143* (Typh.)
Goudoté, E., (359)* (Der.)
Gounelle, H., 557 (Def. Dis.)
Govil, K. K., 1272* (Haem.)
Gozzi, E., 440*, 612* (Am.)
Grabowska, M., 736 (Hel.)
Gracheva, L. I., 765 (Tox.)
Graham, J. E., 105 (Ent.)
Grainger, W. E., 1118* (Leish.)
Gramiccia, G., 516 (Mal.)
Grant, M. W., 1048 (Def. Dis.)
Gräsbeck, R., 71* (Hel.)
Gratzer, W. B., (762)* (Haem.)
Greenberg, J., 421 (Mal.)
Greenberg, M. S., 877* (Haem.)
Greer, J. W., 741* (Hel.)
Gregor, F., (110) (Ent.)
Greisman, S. E., 289 (Typh.)
Grenet, P., 1063 (Misc. Dis.)
Grenier, P., (988) (Ent.), (1046) (Hel.)
Grewal, M. S., (1012) (Tryp.)
Grewal, R. S., 120 (Mal.)
Griffith, I. V., 762* (Haem.)
Griffiths, B. L., 280 (Tryp.)
Griffiths, J. F., (249) (Misc. Pap.)
Grimm, J. G., 610* (Am.)
Grinstein, M., 651* (Haem.)
Grjébine, A., (988)* (Ent.)
Grobov, A. G., 779 (Ent.)
Grodhaus, G., (636) (Hel.)
Groot, H., 1020 (Y.F.)
Grossberg, S. E., 822 (Der.)
Grossowicz, N., (423) (Tryp.)
Grounds, J. G., 77* (Hel.)
Grumbach, F., 156* (Pl.)
Grun, J., 427* (Leish.)
Guedes, A. da S., 10 (Mal.)
Guerrero, L., 1217* (Mal.)
Guillaume, P., 97* (Misc. Pap.)
Guillén P., J., 446*, 842* (Lep.)
Guimarães, F. N., 252 (Reports, etc.)
Guinn, E., 675 (Lab.)
Guinto, R. S., 1241, 1242* (Lep.)
Guirguis, S., 953 (Hel.)

* *joint author*

Gujral, J. S., 468 (Hel.)
Gunaratna, L. F., (273) (Mal.)
Gunasekara, D. B., 1272* (Haem.)
Gunders, A. E., 56 (Lep.)
Gupta, H. L., 1049* (Def. Dis.)
Gupta, S., 873 (Haem.)
Güralp, N., 749* (Hel.)
Gurri, J., 356* (Tox.)
Gus'kova, V. A., 1016 (Typh.)

H

Haage, H., 183*, 746* (Hel.)
Haas, H. G., (1160) (Hel.)
Haas, V. H., 1026 (Pl.)
Habbu, M. K., 1027* (Pl.)
Habibe, O., 330* (Hel.)
Hackett, C. J., 1137 (Ys.)
Hadar, H., 974 (Vms.)
Hadaway, A. B., 674* (Ent.)
Haddow, A. J., 370, 1285* (Ent.), 781 (Reports, etc.)
Hadjinicolaou, J., 575 (Ent.)
Hadler, W. A., 455 *bis*, 456, 457 (Lep.)
Haenecour, F., 464* (Hel.)
Hafez, M., 575 (Ent.)
Haffkine Institute, 1209 (B.R.)
Hagiwara, S., 775* (Parasit.)
Hairston, N. G., 314, 325 (Hel.)
Halawani, A., 60, 632 (Hel.)
Halcrow, J. G., 43 (Pl.)
Hall, L., 1056 (Haem.)
Hall, L. B., 674*, 1206* (Ent.)
Hall, W. J., 885 (Ent.)
Hallberg, L., 872* (Sp.)
Halpern, B., 723* (Am.)
Halstead, B. W., 679 (B.R.)
Hamada, M., 1159 (Hel.)
Hamilton, P. K., 1251 (Hel.)
Hammerstrom, R. J., 673 (Ent.)
Hammon, W., 252 (Reports, etc.)
Hamon, J., 12, 419*, 1220* (Mal.), 367, (490)*, (667) (Ent.)
Hamperl, H., (1198) (Parasit.)
Hamvas, J. J., 1268* (Hel.)
Hanabusa, J., 361*, 659* (Oph.)
Handa, K. L., 1209* (B.R.)
Haneveld, G. T., 218 (Vms.)
Hanks, J. H., 53, 448, 456, 734* (Lep.)
Hannah, E. E., 199 (Hel.)
Hannoun, C., 713* (Y.F.)
Hansen, J. D. L., 757*, 968* (Def. Dis.)
Hansens, E. J., 371 (Ent.)
Happi, C., 972* (Haem.)
Hara, H., 602*, 603* (Typh.)
Hara, J., (1201) (Ent.)
Harada, N., 447*, 630* (Lep.)
Harinasuta, C., 613 (Am.)
Harney, L., 871 (Def. Dis.)
Harris, W. V., 491 (Ent.)
Harrison, J. L., 933 (Typh.)
Harrison, W.O., 319* (Hel.)
Harry, H. W., 63 (Hel.)
Hart, P. de V., 884* (Misc. Dis.)
Harter, P., 1190 (Der.)
Harvey, A. E. C., 816*, 1018* (Typh.), 1118* (Leish.)
Hasbún, R. E., 1178* (Def. Dis.)
Haseeb, M. A., 659 (Heat Str.), 1171* (Hel.)
Hashem, M., 351 (Def. Dis.)
Hashimoto, S., 466* (Hel.)
Hathaway, B. M., 985* (Parasit.)
Hathorn, M., 350*, 867*, 1049*, 1051* (Def. Dis.)
Haug, W. A., 981 (Der.)
Havlík, O., 879 (Tox.)
Hawking, F., 191, 553 (Hel.)
Hayashi, S., 332* (Hel.)
Hayashida, M., (272)* (Mal.)
Hayes, G. R., Jr., 431 (Y.F.)
Hayes, R. O., 368 (Ent.)
Hayes, W. J., Jr., 720 (Pl.)
Hayles, A. B., (563)* (Tox.)
Hechter-Schulz, K., 14 (Mal.)
Hecker, R., (579)* (Misc. Pap.)
Heckly, R. J., 41 (Pl.), 227, 227* (Misc. Dis.)
Hedeen, R. A., 667 (Ent.)
Heeren, J., 55 (Lep.)
Heinz, H. J., 160 (Am.), 1160* (Hel.)
Heisch, R. B., 250, 253 (Reports, etc.), 521, 1118 (Leish.), 698 (Tryp.), 816*, 1018 (Typh.), 960 (Hel.), 1289 (Ent.)
Heller, P., (1056)* (Haem.)
Helluy, J., 1211* (B.R.)
Henderson, A. B., 1182 (Haem.)
Hendrickse, R. G., 87 (Haem.), 511 (Mal.)
Henggeler, C., 859 (Hel.)
Henington, V. M., 204 (Def. Dis.)
Herman, C. M., 702* (Tryp.)
Hermans, E. H., (444) (Lep.)
Hermans, E. H., Sr., (844) (Lep.)
Hermelink, B., 726 (Am.)
Hermosilla, M., (1044)* (Hel.)
Hernandez, A. C., 251 (Reports, etc.)
Hernández, V., 844* (Lep.)
Hernandez Morales, F., (1041)* (Hel.)
Hernández Vallados, R., 331 (Hel.)
Herrer, A., 29 (Bart.), (52) (R.F.)
Hertig, M., 669*, 777* (Ent.)
Hervé, A., 724 (Am.), 1153 (Hel.)
Heuls, J., 219* (Tox.), (979) (Der.)
Hevia, H., 329 (Hel.)
Hewlett, P. S., 1203* (Ent.)
Heyneman, D., 955 (Hel.)
Hezebicks, M. M., 568 (Misc. Dis.)
Hidalgo, R., 521 (Tryp.)
Hiernaux, J., 650* (Haem.)
Higginson, J., 884, 983, (983)* (Misc. Dis.)
Higuchi, K., 825, 1027* (Pl.)
Hilgartner, M., (970)* (Haem.)
Hill, J., 68* (Hel.), (281)* (Tryp.)
Hill, K., 254 (Reports, etc.)
Hill, K. R., 621* (Lep.), 1280 *bis* (Misc. Dis.)
Hilson, G. R. F., 168 (Lep.)
Hinegardner, R. T., 92 (Vms.)
Hirano, N., 56, 845*, 1149 *bis* (Lep.)
Hirose, H., 182*, (328)* (Hel.)
Hitch, J. M., (656) (Der.)
Ho, C., 589 (Mal.)
Hoare, C. A., 237 (Parasit.), 611 (Am.), 926 (Tryp.), 928, 930 (Leish.)
Hocking, B., 578 (Ent.)
Hocking, K. S., 810 (Tryp.)
Hoeke, J. O. O., 218 (Vms.)
Hoekstra, J., 148*, 525*, 605*, 714*, 1122*, 1228*, 1229* (Y.F.)
Hoeprich, P. D., 935 (Typh.)
Hoff, R., 657* (Der.)
Hofshi, E., 881* (Der.)
Hofstra, K., 955 (Hel.)
Hogan, M. J., 978 (Tox.)
Hogerzeil, L. M., 542* (Lep.)
Höglund, N. J., 82* (Sp.)
Hohorst, W., 131* (Mal.)
Holemans, K., 473, 557*, 757, 1049 (Def. Dis.)
Holmes, E. G., 257, 1294 (Reports, etc.)
Holstein, M. H., 256 (Reports, etc.)
Holt, L. E., Jr., 647 (Def. Dis.)
Homawala, K. S., (200) (Def. Dis.)
Hoogstraal, H., 246, (1204), 1289 *bis*, 1290 (Ent.), 255 (Reports, etc.)
Hookins, K. W., 1052* (Haem.)
Hopkins, C. A., (464)* (Hel.)
Hopps, H. E., 814 (Typh.)
Horimoto, T., 1059 (Tox.)
Horn, D. W., 120* (Mal.), 819* (Y.F.)
Horn, H., 646 (Def. Dis.)
Horton, L. L., 651* (Haem.)
Horváth, J., 893* (Ent.)
Hoskins, D. D., 762 (Haem.)
Hoskins, W. M., 1204* (Ent.)
Hosty, T. S., 1125*, 1232 (Rab.)
Hotta, S., 1123 (Den.)
Hourmouziadis, A. N., 673 (Ent.)
House, H. L., (487) (Ent.)
Houston, W. V., 236 (Parasit.)
Hsu, B. R., 799* (Mal.)
Hsü, C. F., 70* (Hel.)
Hsü, H. F., 324 (Hel.)
Hsu, K. C., 799 (Mal.), 326* *bis* (Hel.)
Hsu, M. C., 1043* (Hel.)
Hsü, S. Y. L., 324* (Hel.)
Hu, H. S., 799* (Mal.)
Huang, C. C., 1043* (Hel.)
Huang, C. Y., 1158* (Hel.)
Huang, P. C., 1054 (Haem.)
Huang, S. C., 799* (Mal.)
Huang, W. H., 1043 (Hel.)
Huang, Y. T., 565* (Oph.)
Hubbard, E. D., 866*, 1268* (Hel.)

* *joint author*

Hubbard, J. A., (186)* (Hel.)
Hubbard, J. W., (223)* (Der.)
Hubendick, B., 310, 314* (Hel.)
Hubin, C., 1049* (Def. Dis.)
Hübner, J., 879* (Tox.)
Hudson, A., 488 (Ent.)
Hudson, B. W., 577 *bis* (Ent.)
Huebner, R. J., 253 (Reports, etc.)
Huehns, E. R., 353* (Haem.)
Huet, M., 882* (Oph.)
Huff, C. G., 238 (Parasit.), 806, 808, 1010 (Mal.)
Hughes, J. H., 606 (Y.F.)
Hughes, L. E., 486 (Misc. Dis.)
Hughes, R. D., 1109* (Mal.)
Huldt, G., 481 (Tox.)
Humphries, J. W., 708* (Typh.)
Hunter, R. C., Jr., 359 (Der.)
Huntington, R. W., Jr., 656 (Der.)
Hurley, D. W. H., 1196* (Misc. Dis.)
Hurmusiadis, A., (1164) (Hel.)
Hussain, R., 969 (Haem.)
Husson, R., 135* (Tryp.)
Husson, R. A., 713* (Y.F.)
Hutchison, H. S., 1251* (Hel.)
Huygelen, C., 528, 823 (Rab.)

I

Iablonskaia, V. A., (602)* (Typh.)
Iacovone, R. C., 283* (Tryp.)
Iancu, L., 420* (Mal.)
Iazikov, D. F., 578 (Ent.)
Ickes, C. E., (1216)* (Mal.)
Ihrig, J., 691* (Mal.)
Ikejiri, M., 747* (Hel.)
Ikeshoji, T., 989* (Ent.)
Ikin, E. W., 208* (Haem.)
Ikuzawa, M., 943* (Pl.)
Il'chenko, A. A., 1122* (Typh.)
Ilienko, V., 252* (Reports, etc.)
Imaeda, T., 447*, 630* (Lep.)
Indian J. Dermat., 539 (Lep.)
Ingles, L. G., (1206) (Ent.)
Ingram, V. M., 88* (Haem.)
Inst. Méd. Trop. Princesse Astrid, 998 (B.R.)
Institut Pasteur du Viêt-Nam, 217 (Vms.)
Internat. Nursing Rev., 1293 (Misc. Pap.)
Io, S., (272)* (Mal.)
Ioff, I. G., 609 (Pl.)
Ionesco, N. I., 1231* (Rab.)
Ionescu, H., (935)*, 1227* (Typh.)
Ionescu, I., 745* (Hel.)
Ionescu, N. I., 941*, 1023* (Rab.)
Ionides, C. J. P., 653* (Vms.)
Irie, T., 343 (Hel.)
Irreverre, F., (987) (Ent.)
Isbell, D., 830* (Am.)
Işfan, T., 410*, 420* (Mal.)
Ishak, K. G., 739, 741*, 1154 (Hel.)
Ishihara, S., 949* (Lep.)
Ishii, Y., 747 (Hel.)
Ishizaki, T., 1262 (Hel.)
Israel, M. S., 1267 (Hel.)
Ito, J., (182) (Hel.)
Iuraşog, G., 433* (Rab.)
Iwanaga, S., 479* (Vms.)
Iyengar, M. O. T., 75, 1166 (Hel.)
Izard, Y., 653*, 1273* (Vms.)
Izutani, T., 357 (Tox.)
Izzo, G., (1142) (Lep.)

J

Jackson, E. B., 814* (Typh.)
Jackson, G. J., 805 (Mal.)
Jackson, J. H., 175 (Hel.)
Jacob, G. F., 1180 (Haem.)
Jacob, S., (85)* (Haem.)
Jacobs, L., 93*, 219*, 481*, 655*, (769)*, 1187*, 1189* (Tox.)
Jadassohn, W., 728* (Lep.)
Jadin, J., 253 (Reports, etc.)
Jäger, O. A., 994 (Reports, etc.)
Jame, P., (637)* (Hel.)
James, G. A., 363* (Misc. Dis.)
Jameson, J. L., 269* (Mal.)
Jamison, D. G., 1144 (Lep.)
Jamison, P. W., 1251* (Hel.)
Jampolsky, R., 69* (Hel.)
Jamšek, P., 145* (Typh.)
Jandl, J. H., 877 (Haem.)
Janicki, M., (508) (Mal.)
Jansen, A. A. J., 964 (Def. Dis.)
Janssen, W. A., 437 (Pl.)
Janssens, P. G., 19 (Tryp.), 198 (Hel.)
Janz, G. J., 1114 (Tryp.), 1152 (Hel.)
Jardin, C., 54*, 731* (Lep.), 742* (Hel.)
Jarman, R. T., 1287 (Ent.)
Jaroslow, B. N,. (812) (Tryp.)
Jarpa, A., 162 (Am.)
Jaswant Singh, 414 (Mal.)
Jauregui, J. J., 325* (Hel.)
Jayewardene, L. G., 639* (Hel.)
Jeffery, G. M., 117, 1007 (Mal.), 302 (Am.), 750*, 752* (Hel.)
Jelesić, Z., 1126 (Rab.)
Jelesitsch, Z., 39* (Rab.)
Jelliffe, D. B., (647) (Def. Dis.), 901* (B.R.)
Jellison, W. L., 35* (Den.), 1121* (Typh.)
Jenkins, A. R., 929 (Leish.)
Jensen, D. V., (117)* (Mal.)
Jensen, W. N., 1273* (Haem.)
Jese, L., 1276* (Oph.)
Jeska, E. L., 248 (Lab.)
Jeu, M. H., (1017) (Typh.)
Jevtić, M., 1029 (Am.)
Jezioranska, A., 963 (Hel.)
Jhala, H. I., 1195*, 1196* (Misc. Dis.), 1209 (B.R.), 1230 (Rab.)
Jhala, H. J., (352)* (Haem.)
Jim, R. T. S., 216 (Haem.)
Jiménez-Quirós, O., 170 (Hel.), 830 (Am.)
Jirovec, O., (366), 570, 1066 (Parasit.)
Jöhl, A., (566) (Misc. Dis.)
Johnson, C. M., 1122* (Y.F.)
Johnson, R. E., 362 (Heat Str.)
Jolly, A., 637 (Hel.)
Jolly, S. S., 662* (Misc. Dis.)
Jonchère, H., 135* (Tryp.), 731* (Lep.)
Jones, B., 763 (Haem.)
Jones, F. E., 480, 1189 (Tox.)
Jones, H. L., Jr., 1251* (Hel.)
Jones, J. C., 8, 274, 1221 (Mal.)
Jones, P. R. M., 871 (Def. Dis.)
Jones, S. A., 513 (Mal.)
Jones, W. R., 49 (Am.)
Jonquieres, E. D. L., 843* (Lep.)
Jonsson, U., 973 (Haem.)
Jonxis, J., 254 (Reports, etc.)
Jopling, W. H., 1147 (Lep.)
Jordan, A. M., 1224* (Tryp.)
Jordan, P., 76, 642, 861, 1264 (Hel.), 257 (Reports, etc.)
Joseph, K., 344, 345*, 966 (Def. Dis.)
Josephine, M. A., (Sister), 165 (Am.)
Joubert, S. M., 1052 (Haem.)
Jovanović, L., 1126* (Rab.)
Joyce, B. K., 27* (Typh.)
Juarez, E., 1226* (Leish.)
Juillan, M., 601 (Typh.)
Juminer, B., 550 (Hel.), 562* (Vms.), 799 (Mal.), 1226 (Typh.)
Jung, M., 1276* (Oph.)
Jung, R. C., 1273* (Vms.)
Junnarkar, R. V., 448 (Lep.)
Junnarkar, V. K., 1151* (Hel.)

K

Kaars Sijpesteijn, J. A., 877 (Haem.)
Kabelitz, H. J., 1188 (Tox.)
Kachroo, P., (594) (Mal.)
Kagan, I. G., 240 (Parasit.), 547, 856 (Hel.)
Kahn, E., 968, 1177, 1178 (Def. Dis.)
Kaiser, M. N., 246*, 1290* (Ent.), 255* (Reports, etc.)
Kalabukhov, N. I., 942 (Pl.)
Kalacheva, N. F., 828, 1134* (Pl.)
Kalliomäki, L., 101, 101* (Misc. Dis.)
Kalmykova, A. P., 720* (Pl.)
Kalra, N. L., 117* (Mal.), (667)* (Ent.)
Kalra, R. L., 420* (Mal.)
Kameda, Y., 943* (Pl.)
Kamur, I. B., 429* (Typh.)
Kanai, K., 58* (Lep.)
Kandelwal, M. K., 202* (Def. Dis.)
Kano, R., (1287) (Ent.)
Kao, C. T., 1043* (Hel.)
Kaplan, W., 1191 (Der.)
Karandikar, G. K., 858 (Hel.)
Karani, N. D. P., 576* (Ent.)

* *joint author*

Karcher, D., 19* (Tryp.)
Kariks, J., 662 (Misc. Dis.)
Kark, A. E., 187* (Hel.)
Kartman, L., 533, 610 (Pl.)
Karunaratne, M. D. R., 340* (Hel.)
Karunaratne, W. A., 1218 (Mal.)
Kasliwal, R. M., 614, 834 (Am.)
Kats, F. M., 434* (Rab.)
Kauffmann, M., 1222* (Tryp.)
Kaufman, H. E., 655*, 1187 (Tox.)
Kaul, S., 970 (Haem.)
Kautz, H. D., 206 (Haem.)
Kawachi, S., 479* (Vms.)
Kawagoe, K., 467 (Hel.)
Kawaguchi, M., 288 (Typh.)
Kawaguchi, Y., 58 (Lep.)
Kawamura, A., Jr., 602, 603* (Typh.)
Keegan, H. L., 779 (Ent.), 878 (Vms.)
Keh, B., (636)* (Hel.)
Keiding, J., 670* (Ent.)
Kekcheeva, N. G., 814 (Typh.)
Kelkar, S., 543 (Lep.)
Kellermeyer, R. W., (10)*, (411)* (1216)* (Mal.)
Kelley, G. W., Jr., (1045)* (Hel.)
Kenawy, M. R., 172 (Hel.)
Kendrick, L. P., 421* (Mal.)
Kennedy, B., 204* (Def. Dis.)
Keplinger, M. L., 90* (Vms.)
Kerr, J. A., 1020* (Y.F.)
Kershaw, W. E., 250 (Reports, etc.), 992 (Misc. Pap.)
Kertesz, Z. I., (1176)* (Def. Dis.)
Kettle, D. S., 890 (Ent.)
Key, I. D., 612* (Am.)
Khaikina, A. S., 434* (Rab.)
Khaira, B. S., 884 (Misc. Dis.)
Khajuria, H., (1070) (Ent.)
Khaletskiy, A. M., 764 (Tox.)
Khalil, H. M., 163* (Am.), 460 (Hel.)
Khan, A. A., 661 (Misc. Dis.)
Khanolkar, V, R., 620*, 625* (Lep.)
Khemani, K. M., 1230 (Rab.)
Khin, O., 581* (Reports, etc.)
Kho Lien-Keng, 761 (Haem.)
Khoo Oon Teik, 564* (Der.)
Khundanov, L. E., (159), 299 (Chl.), 720 (Pl.)
Khundanov, L. Y., 720 (Pl.)
Kiefer, E. D., 183* (Hel.)
Kikuchi, H., (186) (Hel.)
Kilejian, A., 1257* (Hel.)
Kilpatrick, J., 676 (Misc. Pap.)
Kilpatrick, J. W., (109)*, 109* (Ent.)
Kimball, A. C., 220, 220* (Tox.)
King, M. A. R., 511* (Mal.)
Kinney, T. D., 473 (Def. Dis.)
Kiossoglou, K., 876 (Haem.)
Kireeva, R. A., 428 (Typh.)
Kirk, R., 222 (Der.), 1045, 1171 (Hel.)
Kirkpatrick, C. I. F., 973* (Haem.)
Kiselev, R. I., 603 (Typh.)
Kissling, R. E., 1125*, 1232* (Rab.)
Kitano, H., 727, 1138 (Lep.)
Kitaoka, M., 151* (Rab.), 934* (Typh.)
Kitselman, C. H., 38 (Rab.)
Kivits, M., 256 (Reports, etc.)
Klein, O., 329* (Hel.)
Klimt, C. R., 171* (Hel.)
Klingberg, W. G., 763* (Haem.)
Klinkhamer, L. V., 598 (Tryp.)
Kloetzel, J., 65*, 743* (Hel.)
Kloetzel, K., 65 *bis*, 743 (Hel.)
Klontz, C. E., 1175* (Def. Dis.)
Kmet, J., 1276* (Oph.)
Knierim, F., 1117 (Tryp.)
Knipe, F. W., 256 (Reports, etc.)
Kobayashi, Y., 1262* (Hel.)
Koblenzer, P. J., 678 (Reports, etc.)
Kobrinskii, G. D., 531* (Rab.)
Koch, A., 602 (Typh.)
Kochwa, S., 771, 1273 (Vms.)
Koeberle, F., 425 (Tryp.)
Kogay, E. S., 687 (Mal.)
Kohls, G. M., 35* (Den.)
Kohn, A. J., 91 (Vms.)
Koike, T., 775* (Parasit.)
Kojima, K., 548* (Hel.)
Kok, J., 218* (Vms.)
Kolesnik, V. S., 720* (Pl.)
Komiya, Y., 326, 548 (Hel.)
Konar, N. R., 443 (Am.)
Kondi, A., 638*, (Hel.) 1053* (Haem.)
Kondo, A., 151, 151* (Rab.)
Kondor, L., 736* (Hel.)
Kono, P., 875* (Haem.)
Konstam, P. G., (1063) (Misc. Dis.)
Kooij, R., 451 (Lep.)
Koppisch, E., 239* (Parasit.), 959 (Hel.)
Kordová, N., 710 (Typh.)
Kordy, M. I., 524 (Typh.)
Korobkova, E. I., 532, 718 (Pl.), 1134 (Chl.)
Körting, H. J., (220) (Tox.)
Kosaka, K., 1149* (Lep.)
Kosminsky, R. B., 719* (Pl.)
Kosobutskii, L. A., (709) (Typh.)
Kotliarevskaya, S. Z., 764 (Tox.)
Kotsinian, M. E., 523 (Typh.)
Kough, R. H., 1056* (Haem.)
Kovaleva, R. V., 826 (Pl.)
Kovalevskii, M. F., 434* (Rab.)
Kowlessar, O. D., (82) (Sp.)
Kozlov, I. S., 287* (Leish.)
Krainova, A. N., 1134* (Chl.)
Krakaur, R. B., (953)* (Hel.)
Král, L., 1126* (Rab.)
Kramer, S., 566* (Misc. Dis.)
Krampitz, H. E., 258 (Reports, etc.)
Krishnamurthy, B. S., 778* (Ent.), 805* (Mal.)
Krishnamurthy, K., (774)* (Misc. Dis.)
Krishnan, K. S., 190 (Hel.)
Krishnan, N. R., 970* (Haem.)
Krudy, A. G., 657 (Der.)
Krylova, O. P., 303* (R.F.)
Kshirsagar, S. G., (648) (Def. Dis.)
Kubik, G., 942* (Pl.)
Kuchukashvili, M. B., 765* (Tox.)
Kugimoto, M., 466* (Hel.)
Kuitunen, E., 1268* (Hel.)
Kulagin, S. M., (429), 430, 711 *bis*, 1121 (Typh.)
Kulikova, V. L., 826* (Pl.), 829* (Chl.)
Kulkarni, V. B., 485* (Misc. Dis.)
Kulonen, E., 101* *bis* (Misc. Dis.)
Kuma, N., 151, 151* (Rab.)
Kumada, N., (246) (Ent.)
Kundu, S., (305)* (Lep.)
Kunert, H., 134* (Tryp.)
Kuntz, R. E., 232, 570, 774 (Parasit.)
Kuo, P. F., 181 (Hel.)
Kupferberg, L. L., 825* (Pl.)
Kuppuswamy, S., 994 (Reports, etc.)
Kurakata, T., 853* (Hel.)
Kurien, P. P., 966 (Def. Dis.)
Kurilova, M. A., 434* (Rab.)
Kurkure, N. B., 620* (Lep.)
Kurzeja, K., 28* (Typh.)
Kusaka, T., 544, (950) (Lep.)
Kuz'mina, A. I., 601* (Typh.)
Kuznetsova, V. I., 1233* (Chl.)
Kvasnicková, D., 411* (Mal.)

L

Laake, E. W., 432 (Y.F.)
Laarman, J. J., 572 (Ent.), 1104 (Mal.)
Labégorre, J., 412* (Mal.)
Labie, D., 215* (Haem.)
LaBrecque, G. C., 490, 893*, 1286, 1290* (Ent.), 803* (Mal.)
Labunets, N. F., 719* (Pl.)
Labzoffsky, N. A., 1268 (Hel.)
Lacan, A., 5 *bis* (Mal.)
Lacaz, C. da S., 223 (Der.), 252 (Reports, etc.)
Lachmajer, J., (6) (Mal.)
Lackman, D. B., 1121* (Typh.)
Lacroix, R., (302)* (Am.)
Ladell, W. S. S., 253 (Reports, etc.)
Ladosky, W., 849 (Hel.)
Lagana, I., (1171) (Hel.)
Lagrange, E., 461 (Hel.)
Laha, P. N., 1195 (Misc. Dis.)
Lahey, M. E., 205 (Def. Dis.)
Lahiri Choudhury, P. K., 44* (Chl.)
Lahitte, F. G., 631 (Hel.)
Laigret, J., 76 (Hel.)
Laing, A. B. G., 551*, 862 (Hel.)

* *joint author*

Lainson, R., 694* (Mal.), 766 (Tox.)
Laird, M., 278, 410 (Mal.), 776 (Parasit.)
Lal, S. B., 472 (Def. Dis.)
Lamberti, J. A., 1260* (Hel.)
Lambotte-Legrand, C., 477*, 876* (Haem.)
Lambotte-Legrand, J., 477, 876 (Haem.)
Lambourg, J., 412* (Mal.)
Lambrecht, F. L., 696*, 697* (Mal.)
Lambrechts, A., 473*, 557, 757*, 1049* (Def. Dis.)
Lämmler, G., 199, 744 (Hel.)
Lamont, N. M., 1051* (Def. Dis.)
Lamont, N. McE., 46 (Am.), 1052 (Def. Dis.)
Lamy, L., (1171) (Hel.)
Lander, H., 1283* (Misc. Dis.)
Lane-Petter, W., 1209 (B.R.)
Langbehn, H. R., 570* (Parasit.)
Languillon, J., 413* (Mal.), 1146* (Lep.)
Lanzkowsky, P., 649 (Haem.)
Lanzo, A., 837 (R.F.)
Lapage, G., 497 (B.R.)
Lapeyssonnie, L., 98 (Misc. Dis.)
Lapierre, J., 20 (Tryp.), 1063 *bis* (Misc. Dis.)
Laranja, F. S., 22* (Tryp.)
Lari, F. A., 278* (Mal.)
Larivière, M., 20*, 134 (Tryp.), 58, 335*, 462 (Hel.), (669)*, 777* (Ent.)
Larizza, P., 761 (Haem.)
Larmane, A., 772* (Oph.)
La Rocca, V., (572) (Ent.)
La Rosa, F., 835 (Am.), 956* (Hel.)
Lashkevich, V. A., (709), 818 (Typh.)
Lathem, W., 1273 (Haem.)
Lau, F. Y. K., (974) (Haem.)
Lauckner, J., 252 (Reports, etc.)
Laugraulet, J., 78* (Hel.)
Laurence, B. R., 469 (Hel.), (889) (Ent.)
Laurent, G., (972), (972)* (Haem.)
Laviron, P., 54 (Lep.)
Lavoipierre, M. M. J., 469 (Hel.), 573 (Ent.)
Lavrenev, B. K., 956* (Hel.)
Law, D. H., (82)* (Sp.)
Lawless, D. K., 232*, 570*, 774* (Parasit.)
Lawy, H. S., 25 (Typh.)
Layrisse, M., 1162*, (1162)* (Hel.)
Le, W. J., 745 (Hel.)
Lea, A. O., (889) (Ent.)
Leal, H., 104* (Ent.)
Learmonth, A. T. A., 376 (Reports, etc.)
Le Breton-Oliveau, G., 658*, 772* (Oph.)
Lechat, M., 541 (Lep.)
Lee, B. K., 746* *bis*, 853*, 1254* (Hel.)
Lee, C. P., 1068* (Ent.)
Lee, J., 799* (Mal.)
Lee, R. D., 197 (Hel.)
Lee Jung Jai, 938* (Rab.)
Leevy, C. M., 867 (Def. Dis.)
Leffkowitz, M., 771* (Vms.)
Lefrou, G., 205 (Def. Dis.)
Le Gac, P., 99 (Misc. Dis.), (108)*, (109) (Ent.), 142* (Typh.)
Legkodimova, K. V., 1118 (Typh.)
Le Golvan, P. C., 739*, 741* (Hel.)
Lehmann, H., 88*, 89*, 208*, 650, 973, (1055), 1272*, (1272)* (Haem.), 254 (Reports, etc.)
Leibina, E. M., 774 (Misc. Dis.)
Leikina, E. S., 956 (Hel.)
Leite, A. S., 252 (Reports, etc.), 1246 (Lep.)
Leithead, C. S., 253 (Reports, etc.)
Leitner, Z. A., 1271 (Def. Dis.)
Lejeune, P., 3 (Mal.)
Lelong, Y., 600* (Leish.)
Lemaitre, Y., (1125)* (Rab.)
Lemon, F. R., 544* (Hel.)
Lemos, F. C., (718) (Pl.)
Leng-Levy, J., (954)* (Hel.)
Lennette, E. H., 655* (Tox.)
Leong Hon Koon, 564 (Der.)
Lépine, P., 1022* (Rab.)
Leppla, W., 484* (Misc. Dis.)
Leprosy in India, 537 (Lep.)
Leprosy Review, 536 (Lep.)
Leshkovich, L. I., 438 (Pl.)
Lessa, A., 282* (Tryp.)
Leszczynski, T., 736* (Hel.)
Le Tan Vinh, 978* (Tox.)
Levaditi, J. C., 135 (Tryp.)
Levi, M. I., 719 (Pl.)
Le Viguelloux, J., 97* (Misc. Dis.), 348* (Def. Dis.)
Levine, H. B., 228* (568), (Misc. Dis.)
Levitus, Z., (46) (Am.)
Levy, A., 1163* (Hel.)
Levy, B., 1227 (Typh.)
Lewallen, L. L., 801, 804 (Mal.)
Lewert, R. M., 239 (Parasit.)
Lewis, D. J., 77 (Hel.), 510 (Mal.), 928, 930, 930* (Leish.), 1286 (Ent.)
Lewis, H. E., 362 (Heat Str.)
Lewis, R. A., 838 (Lep.), 967* (Def. Dis.)
Lewis, W. P., (221) (Tox.)
L'Heureux, M., 524* (Typh.)
Lhoste, J., 943 (Pl.)
Li, C. C., (954)* (Hel.)
Li, H. H., 861 (Hel.)
Li, H. Y., (1159)* (Hel.)
Li, P. S., 324* (Hel.)
Liaskovskaia, Y. I., 720* (Pl.)
Lichtenstein, E. P., 898, 899 (Ent.)
Lichtman, H. C., 206 (Haem.)
Lie-Injo Luan Eng, 876 (Haem.)
Lien, O. G., Jr., 39* (Pl.)
Lien, S. H., 1164 (Hel.)
Lieske, H., 183*, 746* *bis* (Hel.)
Lima, H. M. de A., 1117* (Tryp.)
Lin, C. L., 339* (Hel.)
Lin, L. C., 1045 (Hel.)
Lin, S. S., 463* (Hel.)
Lin, Y. C., 1045* (Hel.)
Linard, R., 1265 (Hel.)
Lindgren, A. G. H., 1044* (Hel.)
Lindgren, I., 1044 (Hel.)
Lindsay, I. S., 578* (Ent.)
Ling, L. H., 627* (Lep.)
Linhard, J., 280* (Tryp.), 650* (Haem.)
Linhart, J., 229* (Misc. Dis.), 280* (Tryp.)
Lippi, M., (136) (Tryp.), (166) (Am.)
Lisbon, 249 (Reports, etc.)
Little, A. M. K., (358) (Tox.)
Littman, M. L., 658* (Der.)
Liu, C. S., 324 (Hel.)
Liu, E. P., 801 (Mal.)
Liu, P. V., 1131 (Chl.)
Liu, T. S., (199)* (Hel.)
Liu, W. T., 341* (Hel.)
Livadas, G. A., 256 (Reports, etc.), 1217 (Mal.)
Livingstone, F. B., 650*, 971* (Haem.)
Llanos, B., 1013* (Tryp.)
Lôbo, J., 1063 (Misc. Dis.)
Loeb, H., 871* (Def. Dis.)
Loianno, R., 296 (Rab.)
Lombardo, G., 881 (Der.)
Lonergan, R. P., 674, 1206 (Ent.)
Longmore, L., 374 (Misc. Pap.)
Lonie, D. A., 1035 (Lep.)
López Antuñano, F. J., (1215) (Mal.)
Losonczi, E., 758 (Def. Dis.)
Lotte, M., 1114* (Tryp.)
Lourmet, J., 742 (Hel.)
Love, G. J., 886 (Ent.)
Lovrić, Š., 145* (Typh.)
Lowenthal, A., 19* (Tryp.)
Lower, E. S., 644* (Hel.)
Lozano Morales, A., 689 (Mal.)
Lozinskaia, T. M., (431)* (Y.F.)
Lu, H. H., 484* (Heat Str.)
Lu, S. C., 1068 (Ent.)
Lu, S. T., (954) (Hel.)
Lubinsky, G., 748 (Hel.)
Luca, E., 433* (Rab.)
Lucas, C. C., 350* (Def. Dis.)
Luder, J., 251 (Reports, etc.)
Ludvík, J., 1187 (Tox.)
Luhar, P. M., 858* (Hel.)
Lukashenko, N. P., 956* (Hel.)
Łukasiak, J., (508)* (Mal.), 1200 (Ent.)
Lum, P. T. M., (889)* (Ent.)
Lumbreras, H., 1013 (Tryp.)
Lumsden, W. H. R., 150 (Den.), 494 (Reports, etc.)
Lunde, M. N., 481, (769), 1189* (Tox.)

* *joint author*

Lundin, F. E., Jr., 841, 1241 (Lep.)
Lunts, L. H. C., 553* (Hel.)
Lunven, P., 412* (Mal.)
Luoto, L., 35* (Den.), (709) (Typh.)
Lupaşcu, G., (269), 1007 (Mal.), 425 (Leish.)
Lusby, M., 228 (Misc. Dis.)
Lutvński, R., 26*, 706, 1120 (Typh.)
Luyken, R., 556, 1048, 1269 (Def. Dis.), 579 (Misc. Pap.)
Luyken-Koning, F. W. M., 1048*, (1269)* (Def. Dis.)
Lyles, S. T., 45 (Chl.)
Lynch, J. E., 444*, 1136 (Am.)
Lýsek, H., 1248 (Hel.)

M

Ma, S. F., 341* (Hel.)
Mabalay, M. C., 1242* (Lep.)
McArthur, J., 1292 (Lab.)
McChesney, T. C., 938 (Rab.)
Macchiavello, A., 155, 436, 1127 (Pl.)
McClelland, G. A. H., 106* (Ent.)
McClelland, W. F. J., 258 (Reports, etc.)
McConnachie, E. W., 618 (Am.)
McCreary, T., 974 (Vms.)
McCulloch, M., 1295 (Reports, etc.)
McCullough, F. S., 1040 (Hel.)
Macdonald, E. M., (77)* (Hel.)
McDonald, W. A., 1223 (Tryp.)
MacDonald, W. E., 90* (Vms.)
Macdonald, W. W., 776 (Ent.)
Macdougall, L. G., 349 (Def. Dis.)
McFadzean, A. J. S., 99 (Misc. Dis.)
McFarland, W., 1182* (Haem.)
McFie, J., 559, 1174* (Def. Dis.)
McGanity, W. J., (1176)* (Def. Dis.)
McGhee, R. B., 279, 697 (Mal.)
McGinniss, G. F., 1288 (Ent.)
Macías, V., 855 (Hel.)
Macintosh, N. W. G., 1181* (Haem.)
MacIver, J. E., 212*, 213, 1183* (Haem.)
McKeever, S., 702 (Tryp.)
McKenzie, D., 649* (Haem.)
Mackerras, M. J., 984 (Parasit.)
McKiel, J. A., 488*, 1285 (Ent.)
Mackinnon, J. E., 770 (Der.)
McLaren, D. S., 349 (Def. Dis.)
McLennan, H., 663* (Misc. Dis.)
MacLeod, J., (490) (Ent.)
McMahon, J. P., 698* (Tryp.)
McMahon, P., 927* (Tryp.)
McManus, A. G., 42* (Pl.)
McMillan, H. L., 366 (Ent.)
MacNab, G. M., 160* (Am.)
Macnamara, F. N., 819 (Y.F.)
McPhee, R., 1289* (Ent.)
Macpherson, R. K., 254 (Reports, etc.)
McRobert, G., 496 (B.R.)
Madeira, O. G., 1114* (Tryp.)
Maeda, M., 949* (Lep.)
Maegraith, B. G., 103 (Parasit.), 256 (Reports, etc.), 276* (Mal.), (579)* (Misc. Pap.), 613* (Am).
Maffi, M., 7, (1105) (Mal.), 632* (Hel.)
Magalhães, A. E. A., 22* (Tryp.)
Magalhães Collaço F., (561)* (Haem.)
Magalhães Neto, B., 65, 68 (Hel.)
Magaudda-Borzì, L., (534)*, 614, 946 (Am.), 1044 (Hel.)
Mahadeva, K., 1272* (Haem.)
Mahfouz, M. S., 244 (Ent.)
Mailhes, N. L., 283* (Tryp.)
Maillot, L., 19, (812) (Tryp.)
Mailloux, M., 55* *bis*, 626*, 731* (Lep.), 336* (Hel.)
Maiorova, I. I., 154* (Rab.)
Majunder, N., 1129* (Chl.)
Malacara, M., 949* (Lep.)
Malakatis, G. M., 570*, 774* (Parasit.)
Malassenet, R., (85)* (Haem.)
Malcolm, S. H., 200*, 472 (Def. Dis.)
Maldonado, J. F., 176, 851, 1267* (Hel.), 1284 (Parasit.)
Maldonado Sampedro, M., 847 (Hel.)
Malek, E. T. A., 250 (Reports, etc.)
Malhotra, M. S., 482 (Heat Str.)
Malossi, M., 235 (Parasit.)
Mamedov, M. M., 956* (Hel.)
Mammen, M. L., 117* (Mal.), 190* (Hel.)
Mammini, P., 1197* (Parasit.)
Manchanda, S. S., 1049 (Def. Dis.)
Mandras, A., 957* (Hel.), 1188* (Tox.)
Maney, W. A. W., 627 (Lep.)
Mangalik, V. S., 873*, 875* (Haem.)
Manigault, P., 1128 (Pl.)
Mann, I., 360 (Oph.)
Mannweiler, E., (949) (Lep.)
Manrique, J. B., 252 (Reports, etc.)
Manson, M. D. E., 658 (Der.)
Manson-Bahr, P., 191, 1263 (Hel.), 373, 492, 992 (Misc. Pap.)
Manson-Bahr, P. E. C., 698* (Tryp.), 928 *bis*, 929* (Leish.)
Mansour, T. E., (1160) (Hel.)
Mansurov, M. N., 273 (Mal.)
Mao, S. P., (69), 327* (Hel.)
Mao, Y. C., 1158 (Hel.)
Maounis, F., 876* (Haem.)
Maqueo, M., 855* (Hel.)
Maranhão, M. A., 959* (Hel.)
Marchal, E., 688 (Mal.)
Marchbank, D. F., 806*, 808* (Mal.)
Marganska, M., 1064* (Parasit.)
Margetts, E. L., 374 (Misc. Pap.)
Margison, J. E., 1136* (Am.)
Marguet, J., 656* (Der.)
Mariani, M., 515* (Mal.)
Mariat, F., 252 (Reports, etc.), 656*, 980* (Der.)
Mariev, A. N., 601 (Typh.)
Marill, F. G., 311, 315, (1041) (Hel.)
Marino, A., (444) (Am.)
Marinone, G., (85) (Haem.)
Mariswamy, M. L., 763 (Haem.)
Markarian, A. G., (429)* (Typh.)
Markell, E. K., (200)* (Hel.), (221)* (Tox.)
Marmion, B. P., 144, (1227) (Typh.)
Marneffe, H., 454 (Lep.)
Maroja, O., 1228* (Y.F.)
Márquez, V., 480* 1060* (Tox.)
Marrero, E., 480*, 1060* (Tox.)
Marroquin, J., 619 (Ys.)
Marsh, C. L., (1045) (Hel.)
Martin, H., 1049* (Def. Dis.)
Martin, J. P., 1016* (Typh.)
Martin, L. A., 769 (Tox.)
Martin, M., 97 (Misc. Dis.)
Martin, W. W. Jr., 1056 (Haem.)
Martin de Mirandol, P., (979) (Der.)
Martinez, P., 1136* (Am.)
Martínez Báez, M., (31) *ter.* (Y.F.)
Martinez Palacios, A., 517 (Mal.)
Martín Jiménez, R., (355) (Tox.)
Martinovic, A., 1008* (Mal.)
Martins, A. R., 257 (Reports, etc.)
Martins, A. V., 310 (Hel.), 1203* (Ent.)
Martins, C. M., 104, 105*, (1202)* (Ent.), 125* (Mal.)
Martins, D., 254 (Reports, etc.)
Maruashvili, G. M., 285 (Leish.), 764 (Tox.)
Marx, H., (562) (Vms.)
Masako, M., 659* (Oph.)
Masia, G. F., 429*, 1227* (Typh.)
Massonnat, J., 37* (Mal.)
Masterton, J. P., 362* (Heat Str.)
Mastràndreà, G., (166)* (Am.), 235 (Parasit.)
Matewa, V., 939 *bis* (Rab.)
Mathen, K. K., 298* (Chl.)
Mathew, K. K., 812* (Tryp.)
Mathews, J., 1268* (Hel.)
Mathis, W., 803 (Mal.)
Mathur, M. S., 614* (Am.)
Mathurin, J., 704*, 705* (Leish.)
Matilla Gomez, V., (438) (Pl.)
Matossian, R. M., 856* (Hel.)
Matsubayashi, H., 775 (Parasit.)
Matsuda, S., 1149* (Lep.)
Matsumura, T., 1260 (Hel.)
Mattingly, P. F., (368) (Ent.), 992 (Misc. Pap.)
Maurer, R. L., (568)* (Misc. Dis.)

* *joint author*

Mauricio, J. V., (703) (Tryp.)
Mayat, M. G. H., 742 (Hel.)
Mazidis, S. P., 1248 (Hel.)
Mazzacca, G., 824* (Rab.)
Mazzotti, L., 638, 645 (Hel.)
M'Bette, H., 1030* (Am.)
M'Bongue, 1030* (Am.)
Medical Research Council, 96 (Heat Str.)
Medinskii, G. M., 155 (Pl.)
Meduski, J., (525) (Typh.)
Meers, P. D., 820, 1021 (Y.F.)
Mehrotra, R. P., 833* (Am.)
Mehta, R. S., 663 (Misc. Dis.)
Mehta, S. P., 834 (Am.)
Meira, J. A., 1252* (Hel.)
Melamed, A. J., 843 (Lep.)
Meleney, H. E., 830* *bis* (Am.)
Mellanby, K., 243 (Ent.)
Mello, A. L., 1167, 1167* (Hel.)
Melnotte, P., 145 (Typh.)
Melo, A. L., 22* (Tryp.)
Melton, M. L., 655*, 1187*, 1189* (Tox.)
Memória, J. M. P., 9*, 126* (Mal.), 469* (Hel.), (1206)* (Ent.)
Menon, P. B., 576 (Ent.)
Merighi, B., (991) (Ent.)
Merrifield, R. C., 981* (Der.)
Merveille, P., 150* *bis* (Rab.)
Metselaar, D., 1214 (Mal.)
Metz, J., 566 (Misc. Dis.)
Meyer, B. J., 649* (Haem.)
Meyer, H., 137 (Tryp.)
Meyer, L. M., 1055* (Haem.)
Meyus, H., 837* (R.F.)
Meza, D., 445* (Lep.)
Mgaloblishvili, O. V., 765 (Tox.)
Michard, V., 205* (Def. Dis.)
Micheloni, F., 664* (Parasit.)
Mickelsen, O., 122* (Mal.)
Micks, D. W., 244*, 370 (Ent.)
Micuda, J., (223)* (Der.)
Miele, E., (444)* (Am.)
Mijares, M. S., 1260 (Hel.)
Mika, L. A., 293, 712, 712*, (Typh.)
Mikata, T., 775* (Parasit.)
Mikeš, A., 1180 (Def. Dis.)
Mikhaleva, V. I., 720* (Pl.)
Mikol, C., 167 (Ys.)
Mikuni, R. T., 107* (Ent.)
Milhade, J., 97*, 101* (Misc. Dis.)
Millemann, R. E., 953 (Hel.)
Miller, D., (886) (Ent.)
Miller, D. J., 154* (Pl.)
Miller, H., 782 (Reports, etc.)
Miller, M. J., 471 (Hel.)
Minkenhoff, J. E., 142 (Typh.)
Minning, W., 632 (Hel.)
Minoda, R., 659* (Oph.)
Minsky-Kravetz, B., 742 (Hel.)
Miquel, R., 628 (Lep.)
Mironov, N. P., (718) (Pl.)
Mîrza, L., 1025* (Rab.)
Mishchenko, N. K., (429)*, 934* (Typh.), (946) (R.F.)
Miškov, D., 1120 (Typh.)
Miskus, R., 1204* (Ent.)
Misra, B. G., 596*, 1106* (Mal.)
Missirliu, C., 1120 (Typh.)
Missirliu, M. F., 1120* (Typh.)
Mitereva, V. G., (522) (Typh.)
Mititelu, P., 1007* (Mal.)
Mitsui, Y., 361, 659 (Oph.)
Miura, N., 1140* (Lep.)
Miura, U., 466 (Hel.)
Mizuno, S., 934* (Typh.)
Möbest, H., 433, 823, 1026 *bis*, 1231 (Rab.)
Moçambique, 1112 (Tryp.)
Mochtar, R., 255 (Reports, etc.)
Modi, C. J., 866* (Hel.)
Modica, R., 816 (Typh.)
Mohammed, A. H. H., 421 (Mal.)
Mohiuddin, A., 813 (Leish.)
Mohr, C. O., 290 (Typh.), 1288 (Ent.)
Mohr, E., (1028)* (Chl.)
Mohr, W., 358* (Tox.)
Moldenschardt, H., 1256* (Hel.)
Molina, J. J., 1066* (Parasit.)
Molinengo, L., 622* (Lep.)
Mondal, A., 44* (Chl.)
Money, G. L., 868 (Def. Dis.)
Mongam, E. S., 88 (Haem.), 359 (Der.)
Monpère, H., 668* (Ent.)
Monson, W. B. L., 677 (Reports, etc.)
Montel, M. L. R., 839 (Lep.)
Montenegro, M. R., 1252* (Hel.)
Montestruc, E., 308 (Hel.), 623, 629, 1034 (Lep.)
Montgomery, J., (974) (Vms.)
Montoya, J. A., 253 (Reports, etc.)
Moon, A. P., 182 (Hel.)
Moon, C. H., 853* (Hel.)
Moore, A. R., 258 (Reports, etc.)
Moore, C. V., 651* (Haem.)
Moore, J. C., 1192* (Der.)
Mooser, H., 51 (R.F.)
Morales, J., (52)* (R.F.)
Mora Lopez, J., (1160) (Hel.)
Morehouse, W. G., 750*, 752* (Hel.)
Morel, P., (991)* (Ent.)
Morelj, M., 604*, 710 (Typh.)
Morgan, H. V., 222* (Der.), 1171* (Hel.)
Mori, R., 951* (Lep.)
Morilleau, R., (637)* (Hel.)
Morlan, H. B., 1288* (Ent.)
Morris, G. C., Jr., 109* (Ent.)
Morris, J. A., 1139 (Lep.)
Morrissey, L. P., 1268* (Hel.)
Mortelmans, J., 528*, 823* (Rab.)
Moser, L., 1067 (Parasit.)
Mosettig, E., 927* (Tryp.)
Mosna, E., 11, 692 (Mal.)
Mosolova, O. N., 829* (Chl.)
Motta, C. M. de P., (1038) (Lep.)
Motta Montes, A., 336* (Hel.)
Mouchet, J., 700 (Tryp.), (1046)* (Hel.), 1108* (Mal.), (1205)* (Ent.)
Moulin, J., (1180) (Def. Dis.)
Moura Lima, M., 105* (Ent.), 125*, 126* (Mal.), 469*, 1168* (Hel.)
Mourant, A. E., 208* (Haem.)
Mousa, A. H., 741* (Hel.)
Moynihan, I. W., 1019* (Typh.)
Mozley, A., 783 (B.R.)
Msangi, A. S., 170* (Hel.)
Muirhead-Thomson, R. C., 459 (Hel.), 591 (Mal.)
Mukerjee, N., 52 (Lep.)
Mukerjee, S., 944*, 1130 (Chl.)
Mukerji, B., 1209 (B.R.)
Mukherjee, A. M., 1133 (Chl.)
Mukherji, A., 1132 (Chl.)
Mullick, D. N., 1057 (Ep. Dropsy)
Murray, E. S., 1276* (Oph.)
Mussini, M., (1031) *bis* (Am.)
Myers, E. N., 664 (Parasit.)

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Nagano, A., (853) (Hel.)
Nagaty, H. F., 163 (Am.), 231* (Parasit.), 460 (Hel.), 1072* (Reports, etc.)
Nail, J., (954)* (Hel.)
Nair, C. P., 190*, 1170 (Hel.)
Nakagawa, P. Y., 107 (Ent.)
Nakamura, K., 1139* (Lep.)
Nakamura, M., 613, 831, 945 (Am.)
Nakamura, S., 695 (Mal.)
Nakase, M., 637 (Hel.)
Nakayama, A., 611 (Am.)
Namba, M., 628* (Lep.)
Nankiewicz, J., 736* (Hel.)
Naquira, F., 357* (Tox.)
Narasimhan, D., 576* (Ent.)
Narayanarao, M., 344* (Def. Dis.)
Nash, T. A. M., 1011, 1224 (Tryp.)
Nataf, R., 772*, 773*, 1061* (Oph.)
Natalizi, G., (576)* (Ent.)
Nath, K., 1281 (Misc. Dis.)
National Schistosomiasis Research Committee, 1041 (Hel.)
Nauck, E. G., 30 (Bart.)
Navarrete, F., 1030* (Am.)
Naylor, R. F., 1245 (Lep.)
Nazimoff, O., 980* (Der.)
Neal, R. A., 251 (Reports, etc.)
Neel, J. V., 650, 971*, 1181* (Haem.)
Negrón, R., 664* (Parasit.)
Nehaul, B. B. G., 648 (Haem.)
Nelson, B. V., 1136* (Am.)
Nelson, G. S., 77, 174, 175, 317, 318, 960* (Hel.)
Nemery de Bellevaux, J., 412 (Mal.)
Neogy, K. N., 1133* (Chl.)
Neri, I., 692* (Mal.)
Nerurkar, R. V., 620* (Lep.)

* *joint author*

Neto, A. V. R., 1141* (Lep.)
Neto, J. A. F., 469 *(Hel.)
Neves, R. G., 455* (Lep.)
Neveu-Lemaire, M., 1210* (B.R.)
Newberne, J. W., 49* (Am.)
Newman, C. G. H., 561 (Haem.)
Newsome, J., 632* (Hel.)
Nguyen Dinh Tiep, 1038* (Hel.)
Nguyen-Tri-Loc, (97)* (Misc. Dis.)
Nguyen-Van-Ai, (97), 773 (Misc. Dis.), 627 *(Lep.)
Nice, P. O., 933 (Typh.)
Nicol, B. M., 558, 755 (Def. Dis.)
Nicol, C. S., 619* (Ys.)
Nicolau, C., 941* (Rab.)
Nicolau, C. S., 1023, 1231 (Rab.)
Nicolau, S. S., (941), 1025, 1231 (Rab.)
Nicolesco, I., (1227) (Typh.)
Niedmann, G., 357* (Tox.)
Nielsen, L. T., 105* (Ent.)
Niemegeers, K., 809 (Tryp.), 896* *bis* (Ent.)
Nigg, C., 227 *bis*, 568* (Misc. Dis.)
Nikhinson, I. M., 429 (Typh.)
Nikolitsch, M., 39 (Rab.)
Nikonov, A. G., 1133 (Chl.)
Nilsson, L. R., 82 (Sp.)
Nishi, H., 467* (Hel.)
Nishihara, H., (200)* (Hel.)
Nishimoto, M., 182* (Hel.)
Nishimura, S., 1149 (Lep.)
Nishiura, M., 447*, 630* (Lep.)
Nishiyama, S., 361* (Oph.)
Nitzulescu, V., 111 (B.R.), 885 (Parasit.), 962 (Hel.)
Nivière, J., 145* (Typh.)
Nogueira, A. R., 254 (Reports, etc.), 561* (Haem.), 1116* (Tryp.)
Nohmi, N., 616, 725 (Am.)
Nooman, Z., 739* (Hel.)
Nor El-Din, G., 72 (Hel.)
Norman, L., 702* (Tryp.), 856, 856* (Hel.)
Noskova, E. G., 522* (Typh.)
Noury, M., 715 (Rab.)
Novales, J., 979 (Der.)
Nuñez, V., 1135* (Am.)
Nwokolo, C., 973* (Haem.)
Nyberg, W., 71 (Hel.)

O

Oak, U., 448* (Lep.)
O'Brien, R. D., (1287) (Ent.)
Ochiai, Y., 845* (Lep.)
O'Connor, H., (359) (Der.), 1193 (Ulc.)
Odang, O., 761* (Haem.)
Oettlé, A. G., (780)* (Ent.), (983), 983* (Misc. Dis.)
Ogata, S., 659* (Oph.)
Ogg, J. E., 40 (Pl.)
Ogura, K., 882 (Oph.)
Ogura, M., 934* (Typh.)
Ohtawara, M., 934* (Typh.)
Ohtsuka, T., (853)* (Hel.)
Oka, M., 943* (Pl.)
Okabe, K., 1253 (Hel.)
Okano, M., 529* (Rab.)
Okpala, I., 807 (Mal.)
Olavarrieta, S. T., 1055* (Haem.)
Oleś, A., 27, 28 (Typh.)
Olesen, E. B., 971 (Haem.)
Olesen, K., 971* (Haem.)
Oliner, H. L., (1056) (Haem.)
Oliveau, 772* (Oph.)
Oliveira, U. A., 1156* (Hel.)
Oliver-González, J., 176*, 460*, 547*, 738*, 850, 959*, (1041), 1267* (Hel.), 239, 1284* (Parasit.)
Olivier, L. J., 64*, 314* (Hel.) 250 (Reports, etc.)
Olmos Castro, N., 450 (Lep.)
Olson, L. J., 77*, 961 (Hel.),
Omardeen, T. A., 517 (Mal.)
Omori, N., 1264 (Hel.)
Ondrácek, J., 1126* (Rab.)
Ono, H., 641 (Hel.)
Onori, E., (343) (Hel.), (1063) (Misc. Dis.)
Oomen, H. A. P. C., 200, 556, (559), 868 (Def. Dis.)
Ootaka, K., 1140 (Lep.)
Oppenoorth, F. J., 778* (Ent.)
Ordman, D., 771 (Vms.)
Ordóñez Acuña, A., (860)* (Hel.)
Orfila, J., 130*, 595*, 806* (Mal.)
Orio, J., 150, 156* (Rab.), 219, 977 (Tox.), (979)* (Der.)
Orlandella, V., 1274* (Tox.)
Orlov, G. A., 764, 765 (Tox.)
Orlova, N. N., 940 (Rab.)
Orłow, G., 768 (Tox.)
Orłowska, B., (525)* (Typh.)
Ormerod, W. E., 519 (Tryp.)
Osada, M., 1235 (Am.)
Osada, Y., 428* (Typh.)
Osimani, J. J., (747) (Hel.)
Otsuji, K., 466* (Hel.)
Otto, G. F., 231 (Parasit.)
Ovazza, M., (989) (Ent.)
Oxenstein, A., 725* (Am.)
Oytun, H. S., 749 (Hel.)
Ozawa, T., 466* (Hel.)

P

Pacheco de Figueiredo, J. M., 1197 (Parasit.)
Paciorek. H., 736* (Hel.)
Packchanian, A., 284, 1222 (Tryp.)
Padaria, F. T., 834* (Am.)
Padilha, C., 139* (Tryp.)
Pagnotta, G., (330)* (Hel.)
Pagon, S., 604* (Typh.)
Paillet, R. J., (1164)* (Hel.)
Paine, D. H. D., 644 (Hel.)
Palmer, J. R., 460* (Hel.)
Palmieri, C., 692* (Mal.)
Pampana, E. J., 257 (Reports, etc.)
Pampiglione, S., 224 (Oph.), 555 (Hel.), 778 (Ent.), 975 (Vms.)
Panaitesco, D., 335*, 962* (Hel.)
Pandeye, S. N., 1281* (Misc. Dis.)
Panja, G., 54 (Lep.)
Pannarale, R., 882 (Oph.)
Panthier, R., 526, 713 (Y.F.)
Papadakis, A. M., 673 (Ent.)
Papastamatiou, J., 86* (Haem.)
Paraense, W. L., 180, 1250 (Hel.)
Paranjpey, P. D., 751* (Hel.)
Paravissini, F., 1267 (Hel.)
Parekh, J. G., (352)* (Haem.), 485 (Misc. Dis.)
Paretsky, D., 27 (Typh.)
Parfitt, J. W., 366 (Parasit.)
Parish, J., 890* (Ent.)
Parish, R. H., 890* (Ent.)
Parmelee, W. E., 197* (Hel.)
Parodi, L., 301, 724* (Am.)
Paronikyan, G. M., 704 (Leish.)
Parr, H. C. M., 895 (Ent.)
Parrish, H. M., 879 (Vms.)
Parrot, L., (490) (Ent.)
Parys, G., 475 (Def. Dis.)
Pascu, A., 754 (Hel.)
Pasmanik, S., (857)* (Hel.)
Pastukhova, G. M., 706 (Typh.)
Patel, B. D., (352) (Haem.), 485* (Misc. Dis.), 967* (Def. Dis.)
Patel, J. C., 617* (Am.)
Patel, M. V., 858* (Hel.)
Patel, S. M., 1209* (B.R.)
Patel, T. B., 751 (Hel.)
Paterson, J. C. S., 1056 (Haem.)
Paterson, S., 461* (Hel.)
Pattoli, D., 988* (Ent.)
Patwardhan, V. N., (648)*, 1177* (Def. Dis.), 969* (Haem.)
Patzelt, O., 200 (Hel.)
Paulini, E., 126,* 412*, 413 (Mal.), 256 (Reports, etc.), (321)*, 322* (Hel.), (1206) (Ent.)
Pavilanis, V., 524 (Typh.)
Pavlovsky, J. N., 993 (Misc. Pap.)
Pawłowski, Z., (1196) (Parasit.)
Payet, M., 228, 229 (Misc. Dis.)
Pearlstein, H., 664* (Parasit.)
Pearson, H. A., 971, 1182 (Haem.)
Pecchiai, L., 571 *bis* (Parasit.)
Pech, J., (1202)* (Ent.)
Peel, E., 5*, 697* (Mal.), 700* (Tryp.)
Pelikán, V., 411 (Mal.)
Pellegrino, J., 286* 600 (Leish.), 316, 547, 636*, 1156 *bis*, 1250 (Hel.), 1117* (Tryp.), 1203* (Ent.)
Pène, P., 228*, 229* (Misc. Dis.)
Pennisi, L., 614*, 946* (Am.), 1044* (Hel.)
Pepler, R. D., 483 (Heat Str.)
Pepler, W. J., 235 (Parasit.)
Pepys, J., 252 (Reports, etc.)
Perederi, S., (935)*, 1227* (Typh.)

* *joint author*

Pereira, J. P., 412*, 413* (Mal.)
Pereira, O., 180* (Hel.)
Pereira, S., 833* (Am.)
Pérez-Giménez, M. E., (1162)*, 1162*, 1163* *bis* (Hel.)
Pérez-Miravete, A., 948 (Ys.)
Pérez-Moreira, L., 356* (Tox.)
Pérez Pérez, B., 622* (Lep.)
Pérez Reyes, R., 689* *bis* (Mal.)
Périsse, J., (557) (Def. Dis.)
Perlina, F. I., (153) (Rab.)
Perna, V. P., 465* (Hel.)
Perry, A. S., 896 (Ent.)
Pesigan, T. P., 325 (Hel.)
Pessoa, S. B., 251 (Reports, etc.), 380 (B.R.), 320, 1150 (Hel.)
Pestel, M., 627* (Lep.)
Peters, J. A., 81* (Def. Dis.)
Peters, W., 269, (1106), 1110 (Mal.)
Peterson, D. G., 574*, (889)* (Ent.)
Petrescu, A., 1025* (Rab.)
Petrides, J., 516* (Mal.)
Petrischeva, P. A., 889 (Ent.)
Petrov, A. A., 523* (Typh.)
Petrova, B. I., 1134* (Chl.)
Pétrovitch, Z., 767*, 768* (Tox.)
Pezzullo, C., 615 (Am.)
Pham Kim Luong, 1038 (Lep.)
Phan-Ngoc-Duong, (97)* (Misc. Dis.)
Philip, C. B., 486* (Misc. Dis.)
Phillips, B. P., 723 (Am.)
Philpot, V. B., Jr., (1057) (Vms.)
Pickens, E. G., 35 (Den.)
Piedrola Gil, G., (438)* (Pl.)
Pierce, D. E. M., 763* (Haem.)
Pierquin, L., 896 *bis* (Ent.), 998 (B.R.)
Piffaretti, M. L., (353)* (Haem.)
Piganiol, G., 724* (Am.), 1153* (Hel.)
Pike, E. H., 1261 (Hel.)
Pille, G., 412 (Mal.)
Pinkhas, J., 352* (Haem.)
Pinotti, M., 256 (Reports, etc.)
Pinto, A., 1246* (Lep.)
Pinto, C., 703 (Tryp.)
Pinto Severo, O., 527 (Y.F.)
Piontkovskaia, S. P., 934 (Typh.)
Piper, D. W., (476) (Sp.)
Pipkin, A. C., (117) (Mal.)
Piquett, P. G., 490* (Ent.)
Pires, C. D. de A., 165* (Am.), 1151* (Hel.)
Pires, W. M., 1168* (Hel.)
Piringer, W. A., 336 (Hel.)
Pirkle, C. I., 122* (Mal.)
Pirsch, J. B., 293*, 712, 712* (Typh.)
Pitchford, R. J., 60, 307, 319, 546* (Hel.)
Pitman, C. R. S., 654* (Vms.)
Pitzschke, H., 715 (Rab.)
Piva, F., 664* (Parasit.)
Pizzolato, P., 98 (Misc. Dis.)
Placidi, L., 1230 (Rab.)
Planchet, L., 622 (Lep.)
Platt, B. S., 253 (Reports, etc.), 346, 557 (Def. Dis.)
Pletnikova, G. P., 720* (Pl.)
Poirier, A., (953) (Hel.)
Polak, M., 1252 (Hel.)
Polish Parasitol. Soc., 569 (Parasit.)
Politzer, W. M., 111 (Misc. Pap.), 758 (Def. Dis.)
Pollard, C. B., 879* (Vms.)
Pollard, D. G., 273* (Mal.)
Pollitzer, W. S., 651 (Haem.)
Pollok, N. L., 293*, 712* (Typh.)
Polman, A., 976 (Tox.)
Polunin, I., 377 (Reports, etc.)
Pomerantzev, B. J., (991) (Ent.)
Pompeu Memoria, J. M., 1250* (Hel.)
Pontes, J. F., 1252* (Hel.)
Pooler, N. R., 46* (Am.)
Popa, S., (1227)* (Typh.)
Pop-Cénitch, S., 767*, 768* (Tox.)
Pope, J. H., 1019* (Typh.)
Popova, L. D., 1121* (Typh.)
Popović, B., 754 (Hel.)
Portal, A., (637)* (Hel.)
Porte, L., (1121) (Typh.)
Porter, J. E., 606* (Y.F.)
Porter, R. J., 159* (Am.)
Porterfield, J. S., 819* (Y.F.)
Portilla, J., 548*, 551* (Hel.)
Postmus, S., 556, 1049 (Def. Dis.)
Potter, E. L., (970) (Haem.)
Potter, M. D., 553* (Hel.)
Potts, W. H., 597 (Tryp.)
Povolný, D., (110)* (Ent.)
Powell, D. E. B., 818* (Typh.)
Powell, S. J., 759 (Haem.), 444*, 832, 1236, 1237 (Am.)
Prades, C. M., 130* (Mal.)
Prankerd, T. A. J., 353* (Haem.)
Prasad, B. G., 1272* (Haem.)
Pratt, J. J. Jr., 373* (Ent.)
Press, J. M., 898 (Ent.)
Preston, H., 942* (Pl.)
Pretorius, H. M., 1219* (Mal.)
Pretorius, P. J., 348*, 474, 966 (Def. Dis.)
Price, E. W., 445, 729, 1143 (Lep.)
Price, R. B., 1035 (Lep.)
Prieto Lorenzo, A., (241) (Ent.), (641) (Hel.)
Prince, F. M., 533* (Pl.), 577* *bis* (Ent.)
Priolet, G., 731* (Lep.)
Proc. Nutrition Soc., 362 (Heat Str.)
Proctor, N. S. F., 1160* (Hel.)
Proreshnaia, T. L., (429) (Typh.)
Protti, P., 975 (Vms.)
Provost, A., 1023* (Rab.)
Przyborowski, T., 532* (Pl.)
Pshenichnov, A. V., 522 (Typh.)
Purcell, M. K., 87* (Haem.)
Purser, J. R., 847 (Hel.)
Pyne, C. K., 703 (Leish.)

Q

Quagliato, R., 1036 (Lep.)
Quan, S. F., 42, 533* (Pl.)
Quattrocchi, G., 956 (Hel.)
Qutubuddin, M., 988 (Ent.)

R

Raba, A., 58* (Hel.)
Rabello, E. X., 835* (Am.), 988* (Ent.)
Rachou, R. G., 105, (1202) (Ent.), 125, 126 (Mal.), 338, 339, 469, 470, 1167* *bis*, 1168, 1169 (Hel.)
Radacovici, E., 410*, 420, 1007 (Mal.)
Radermecker, J., 198* (Hel.)
Radermecker, M. A., 557* (Def. Dis.)
Radnot, M., 981 (Oph.)
Radomski, J. L., 89, 90* (Vms.)
Radovanovic, M., 648* (Def. Dis.)
Radvan, R., 604* (Typh.)
Rafi, S. M., 685 (Mal.)
Ragab, H. A. A., 595 (Mal.)
Rageau, J., 1183* (Vms.)
Raghavan, N. G. S., 190* (Hel.)
Raginis, Z., 26*, 1120* (Typh.)
Rahm, U., 244 (Ent.)
Rahman, J., 594 (Mal.)
Rai, L., 892 (Ent.)
Raigorodskaia, V. I., 578* (Ent.)
Rainaut, J., 280 (Tryp.)
Rajan, K. T., 1282* (Misc. Dis.)
Rajenison, S., 435* (Pl.)
Rakotomanga, J. C., (1125)* (Rab.)
Rakotomihamina, S., 435* (Pl.)
Ramakrishna, V., 419*, 804 (Mal.)
Ramakrishnan, C. V., 646* (Def. Dis.)
Ramakrishnan, S. P., 414* (Mal.)
Rama Rao, R., 17 (Mal.)
Ramirez, M., 1173* (Hel.)
Ramoo, H., 468* (Hel.)
Ramos, A. da S., 1117 (Tryp.)
Ramos, J. R., 445* (Lep.)
Ramos e Silva, J., (949) (Lep.)
Ramsdale, C. D., 419* (Mal.)
Ramu, G., 1142 (Lep.)
Ranadive, K. J., 543*, 620 (Lep.)
Randriamalala, J. C., 1153* (Hel.)
Ranney, H. M., 875 (Haem.)
Ranque, J., 704*, 705 (Leish.)
Rao, B. A., 414* (Mal.)
Rao, B. R. H., 964, 1175 (Def. Dis.)
Rao, G. S., 967* (Def. Dis.)
Rao, K. S., 1177 (Def. Dis.)
Rao, K. V., 444 (Am.)
Rao, M. V. R., 1209 (B.R.)
Rao, P. S. S., 963*, 1175* (Def. Dis.)
Rao, R. S., 1209 (B.R.)
Raoult, A., 963, 1270 (Def. Dis.)

* *joint author*

Raper, A. B., 592 (Mal.), 1062 (Misc. Dis.)
Rasmussen, H., 647* (Def. Dis.)
Rasooly, G., (423)* (Tryp.)
Rateau, J., 700* (Tryp.)
Ratgauz, V. G., 154* (Rab.)
Rathburn, C. B., Jr., 108* *bis* (Ent.)
Rathi, M. M., 834* (Am.)
Rathscheck, H. J., 921 (Mal.)
Ratovondrahety, (1191)* (Der.)
Ratulangie, C. J. A., 630* (Hel.)
Raud, J., 731* (Lep.)
Ravisse, P., 969 (Haem.), 977* (Tox.)
Rawal, B. D., 563, 766, 977 (Tox.)
Ray, A. P., 126 *bis*, 596* *bis*, 1106 (Mal.)
Ray, R. N., 1181* (Haem.)
Razafimandimby, C., 435* (Pl.)
Razumeenko, T. V., 155* (Pl.)
Ré, L., 1246* (Lep.)
Read, C. P., 237 (Parasit.), (344)* (Hel.)
Read, T. R., 1236* (Am.)
Rees, D. M., 105* (Ent.)
Reeves, R. E., 830 *bis* (Am.)
Reeves, W. C., 368* (Ent.)
Rêgo, S. F. M., 22 (Tryp.)
Rehm, W. F., 515, 1107* (Mal.)
Rehn, F., 29, 431, 604 (Typh.)
Reid, J. A., 15 (Mal.), 191*, 551* (Hel.), 1290 (Ent.)
Reif, A., 1198 (Parasit.)
Relvich, A. L., 947, 1032 (Ys.)
Remington, J. S., 655, 1187* (Tox.)
Renoux, G., 562 (Vms.), 772, 772*, 773* *bis*, 1061 (Oph.)
Reutner, T. F., 130* (Mal.)
Revelles, J. P., 453* (Lep.)
Rey, L., 329 (Hel.)
Reyes G., G., 705 (Leish.)
Reynaud, R., 214* (Haem.), 647* (Def. Dis.), 957*, 1163* (Hel.)
Rhodes, F. A., 565*, (1195)* (Misc. Dis.)
Rhodes, K., 1280 (Misc. Dis.)
Ribeiro, A. M., 51 (Am.)
Ribeiro, J. A. A. C., 253 (Reports, etc.)
Ribeiro do Rosário, M., (561) (Haem.)
Ricci, M., 234, 365, 1064 (Parasit.)
Ricciardi, M. L., 440* *bis*, 612, 724* (Am.)
Rice, C. E., 1019* (Typh.)
Rice Inst. Pamphlet, 236 (Parasit.)
Richert, J. H., (953)* (Hel.)
Richet, P., 1114 (Tryp.)
Richter, L., 620 (Lep.)
Rifaat, M. A., 163* (Am.), 231 (Parasit.), 460* (Hel.)
Rigaud, J. L., 1030* (Am.)
Rijpstra, A. C., 439* (Am.)
Rimbert, C. L., (82)* (Def. Dis.)
Rimini, R., (465)* (Hel.)
Rioux, J. A., 270 (Mal.), (1202) (Ent.)
Rishikesh, N., (591) (Mal.)
Ritchie, L. S., 233* (Parasit.)
Rivas-Gomez, A., (1164) (Hel.)
Rivoalen, A., 97*, 101* (Misc. Dis.)
Rivosecchi, L., 11*, 692* (Mal.), (109), 890* (Ent.)
Roan, C. C., 245*, 892* (Ent.)
Roath, O. S., 973* (Haem.)
Robertaccio, A., (444)* (Am.)
Roberts, A. N., 709 (Typh.)
Robertson, A. G., 700 (Tryp.)
Robertson, D. H. H., 929* (Leish.)
Robertson, E. G., 1283* (Misc. Dis.)
Robinson, A. R., 284* (Tryp.), 650*, 971*, 1181* (Haem.)
Robinson, D. L. H., 632* (Hel.)
Robinson, E. J., Jr., 1157 (Hel.)
Robinson, P., 363 (Misc. Dis.)
Robinson, S., 253 (Reports, etc.)
Robledo, E., 1039* (Hel.)
Robles Garnica, R., 428 (Typh.)
Robson, H. N., 565*, (1195)*, 1283* (Misc. Dis.)
Robson, J., (423) (Tryp.)
Robson, J. M., 734 (Lep.)
Roca Estevez, A., (465)* (Hel.)
Roch, E., 480*, 1060* (Tox.)
Roche, M., 1162, (1162), 1163 *bis* (Hel.)
Rockenmacher, M., 41* (Pl.)
Rodenwaldt, E., 902 (B.R.)
Rodger, F. C., 865 (Hel.)
Rodman, T., 87* (Haem.)
Rodrigues da Silva, J., 24, 140, 286, (427) (Leish.), 48, 49 (Am.), 67 (Hel.)
Rodriguez, H., 344* (Hel.)
Rodriguez, J. D., (634)* (Hel.)
Rodriguez, J. N., 844*, 1244 (Lep.)
Rodriguez-Molina, R., 351* (Sp.), 738 (Hel.)
Rodríguez Neto, A., (353)* (Haem.)
Rodriquez, G., (330)* (Hel.)
Roels, O. A., 253 (Reports, etc.)
Roger, A., 141*, 1017* (Typh.)
Roger, F., 141, 1017 (Typh.)
Rogers, A. J., 108 *bis* (Ent.)
Rogers, E. W., 640 (Hel.)
Rogozina, E. N., 154* (Rab.)
Rohde, K., 961 (Hel.)
Roitman, E., 647* (Def. Dis.)
Rojas, C., 1173 (Hel.)
Rollier, R., (82) (Def. Dis.)
Roman, E., (641) (Hel.)
Romaña, C., 138, 139 (Tryp.), (355) (Tox.)
Roman y Carrillo, G., 416* (Mal.)
Römer, M. A., 1055 (Haem.)
Romero Jordán, O., 1276* (Der.)
Romiti, C., 119 (Mal.)
Rosen, L., 33, 34 *bis* (Den.)
Rosen, S. W., 183 (Hel.)
Rosenbaum, M. B., (424) (Tryp.)
Rosický, B., (366)* (Parasit.)
Rosillo, J., 352* (Haem.)
Ross, H., 728*, 841*, 1241* (Lep.)
Rothenberg, S. P., (1258)* (Hel.)
Rothstein, N., 775 (Parasit.)
Roubaud, E., (1068) (Ent.)
Roullier, C., 965* (Def. Dis.)
Rousset, J. J., 20* (Tryp.)
Rowan, W. B., 176 (Hel.)
Roy, N., 726* (Am.)
Roy, R., 1170* (Hel.)
Royal Soc. Promotion Health J., 611 (Am.), 676 (Misc. Pap.)
Roychowdhury, S. P., 472* (Def. Dis.)
Royol, J., 356* (Tox.)
Rozeboom, L. E., 106, 367 (Ent.), 937 (Den.)
Rozhkov, F. V., 819* (Typh.)
Rubin, M. L., 766 (Tox.)
Rubin, R., 283* (Tryp.)
Rudra, B. C., 944* (Chl.)
Ruffié, J., 16*, 270* (Mal.), 282* (Tryp.), (1204)*, 1204* (Ent.)
Ruggiero, C., 124* (Mal.)
Ruiz, A., 813 (Tryp.), 835 (Am.)
Ruiz, J. M., (64) (Hel.)
Rumiantseva, E. V., 819* (Typh.)
Rundkvist, V. A., 578* (Ent.)
Russell, D. A., 1240 (Lep.)
Russell, H. B. L., 255 (Reports, etc.)
Russell, P. F., 236 (Parasit.)
Rutgers, A. W. F., 451* (Lep.)
Rüttner, J. R., (1160)* (Hel.)
Ruzette, M. A., (1206)* (Ent.)

S

Sabin, A. B., 252 (Reports, etc.)
Sabour, M., 739* (Hel.)
Saccà, G., 890, 891, 895 (Ent.)
Sachdev, S., 163, 833 (Am.)
Sadek, G., 60* (Hel.)
Sadun, E. H., 463 *bis*, 746* *bis*, 852, 853, 856*, 1254 (Hel.)
Saf'yanova, V. M., 889* (Ent.)
Sagone, M., 164 (Am.)
Saha, T. K., 726* (Am.)
Saif, M., 616* (Am.)
Sakamoto, Y., 182*, (328)* (Hel.)
Sakanashi, M., 361* (Oph.)
Salamon, T., 203, 648 (Def. Dis.)
Salas, M., 441 (Am.)
Salas Panisello, F., (355)* (Tox.)
Salem, S., 163* (Am.)
Sales, S., 12* (Mal.), 367* (Ent.)
Salib, M., 739* (Hel.)
Salunke, D. S., 858* (Hel.)
Salvi, S., (1207)* (Ent.)
Samarawickrema, W. A., 245* (Ent.)
Sameshima, H., (470) (Hel.)
Samios, C. J., 512 (Mal.)
Sampaio, S. A. P., 223* (Der.)
Sams, W. M., 1266 (Hel.)

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Sánchez Domínguez, A., 38 (Rab.)
Sandbank, A., 1198* (Parasit.)
Sandberg, E., 464 (Hel.)
Sandosham, A. A., (58) (Hel.), 1298 (B.R.)
Sandulesco, M., 593* (Mal.)
Sanghvi, L. D., 89 (Haem.)
Sankalé, M., 97, 101 (Misc. Dis.), 348 (Def. Dis.)
Sankaran, A. N., 345* (Def. Dis.)
Sanmartin, C., 1020 (Y.F.)
Sansarricq, H., 1248 (Hel.)
Santillan, G., 445* (Lep.)
Santoro, E., 1274* (Tox.)
Santos, A., 1246* (Lep.)
Santos, A. T., 325* (Hel.)
Santos, B. C., 325* (Hel.)
Santos, D., (339)* (Hel.)
Santos, U. M., 600* (Leish.)
Saraiya, C. C., (352)* (Haem.)
Sarasin, G., 166* (R.F.)
Sargent, F., 362* (Heat Str.)
Sarin, B. P., 560 (Def. Dis.)
Sarkar, J. K., 157 (Chl.)
Sasa, M., 110, 246 (Ent.), 143, 428 (Typh.), 332 (Hel.)
Satake, Y., 1145 (Lep.)
Satani, Y., 732 (Lep.)
Sati, M. H., 251 (Reports, etc.)
Sato, S., 843 (Lep.)
Sato, T., (470)* (Hel.)
Sato, Y., 1259 (Hel.)
Satoo, J., 1140* (Lep.)
Satoo, S., 1140* (Lep.)
Satti, M. H., 1171* (Hel.)
Saunders, D. C., (865) (Hel.)
Saunders, P. R., 879 (Vms.)
Saurat, P., 1063 (Misc. Dis.)
Sautet, J., 368, 369, (1202) (Ent.)
Savchenko, E. N., 429* (Typh.)
Sawyer, T. K., 708* (Typh.)
Sayed, B. A., 984* (Parasit.)
Saz, H. J., (186), (1259) (Hel.)
Scaff, L. M., 1169* (Hel.)
Scarlat, M., 410* (Mal.)
Scatozza, F., 36 *bis* (Rab.)
Schacher, J. F., 337* (Hel.), 1061* (Misc. Dis.)
Schaeffer, M., 1232* (Rab.)
Schaller, J. K., 626* (Lep.)
Schaller, K. F., 1033 (Lep.)
Schao, Y. L., 326* (Hel.)
Schapira, G., 215* (Haem.)
Schapiro, M. M., 1066 (Parasit.)
Schendel, H. E., 757, 968 (Def. Dis.)
Schenone, H., 329* (Hel.), 1058, 1185 (Vms.)
Scherer, W. F., 822* (Den.)
Schiller, E. L., (855), (1043) (Hel.)
Schinazi, L. A., 1257* (Hel.)
Schirmer, E., 1058* (Vms.)
Schmid, A. W., 1022 (Rab.)
Schmid, K., 732 (Lep.)
Schmid, R., 184 (Hel.)
Schmidt, A. M., 935* (Typh.)
Schmidt, C. H., 666, 987* (Ent.)
Schnabel, R., 1256* (Hel.)
Schneider, J., 413 (Mal.), 731 1146 (Lep.), 747 (Hel.)
Schneider, L. E., 293* (Typh.)
Schneider, M. D., 529 (Rab.)
Schneider, R., 119 (Mal.)
Schneider, R. G., 762* (Haem.)
Schnieden, H., 350* (Def. Dis.)
Schofield, F. D., 738 (Hel.)
Schoof, H. F., (109) (Ent.), 803* (Mal.)
Schork, A. R., (1041)* (Hel.)
Schraibman, I. G., 546* (Hel.)
Schramm, G., (1028) (Chl.)
Schreiber, G., 9, 10* (Mal.)
Schrier, S. L., (10)*, (411), (1216) (Mal.)
Schrire, V., 983, 1279 (Misc. Dis.)
Schuler, R. W., 612* (Am.)
Schulman, I., 353*, 652*, (763)* (Haem.)
Schulz, K. H., (200) (Def. Dis.), 712 (Y.F.)
Schulz, K. R., 898*, 899* (Ent.)
Schwabe, C. W., 1257 *bis*, (1258)* (Hel.)
Schwarte, L. H., 81* (Hel.)
Schwartz, C. J., 364 (Misc. Dis.)
Sciarz, W., (26) (Typh.)
Scorza, J. V., 462* (Hel.)
Scott, A., 594, 694* (Mal.)
Scott, D., 255 (Reports, etc.)
Scott, E. M., (365)* (Misc. Dis.), 762 (Haem.)
Scott, J. A., (77) (Hel.)
Scott, R. B., 972* (Haem.)
Scrimshaw, N. S., 205*, (758), (758)*, 966*, 967, 967* (Def. Dis.)
Seal, S. C., 296 (Pl.), 298, (299) (Chl.), 1209 (B.R.)
Segretain, G., 656*, (979)*, 980 (Der.)
Sehgal, B. S., 377 (Reports, etc.)
Seidl, S., 433* (Rab.)
Seki, J., (853)* (Hel.)
Selimov, M. A., 154, 434, 531* (Rab.)
Semenova, E. L., (718) (Pl.)
Semenova, E. V., 434* (Rab.)
Semprevivo, L., 1058* (Vms.)
Sen, N. R., 305* (Lep.)
Sen, P., (8), 272, 420 (Mal.)
Sénécal, J., 965 (Def. Dis.)
Senevet, G., (488), (667), 668, 985 (Ent.), 1104 (Mal.)
Seneviratne, R. D. de A., 1272 (Haem.)
Senft, A. W., 634 (Hel.)
Sen Gupta, G. P., 596 *bis* (Mal.)
Sen Gupta, P. C., 24* (Leish.), 1106* (Mal.)
Sepulcri, P., 408*, 409* (Mal.), 719* (Pl.)
Serbănescu, G., 1007* (Mal.)
Sergent, Ed., 129, 1111 (Mal.)
Sérié, C., 626 (Lep.)
Serrano, D. G., 738* (Hel.)
Service, M. W., 510 (Mal.), (887), (1201) (Ent.)
Sethna, Y. I., 530 (Rab.)
Shafei, A. Z., 1238 *bis* (Am.), 1299 (B.R.)
Shaffer, J. G., 612 (Am.)
Shah, B. N., 866 (Hel.)
Shah, J. R., 834 (Am.)
Shah, U. S., 834* (Am.)
Shang, Y. C., 745* (Hel.)
Shanker, A., 929 (Leish.)
Shaper, A. G., 659 (Misc. Dis.)
Shaper, L., 659* (Misc. Dis.)
Shapiro, M., 210 (Haem.)
Shapiro, M. I., 523*, 1017 (Typh.)
Sharad Kumar, 873*, 875* (Haem.)
Sharma, G. K., 126*, 596* (Mal.)
Sharma, J. C., (594)* (Mal.)
Sharma, K. D., 447 (Lep.), 1275 (Der.)
Sharma, M. I. D., 420, 805 (Mal.), 778 (Ent.)
Sharma, R., 1134 (Am.)
Shatrov, I. I., 932 (Typh.)
Sheldon, W. H., 885, 1199 (Parasit.)
Shelley, W. B., 74* (Hel.)
Shen, J. S., 1158* (Hel.)
Sheng, S. C., 1158* (Hel.)
Shenoy, M. A., 873* (Haem.)
Shepard, C. C., 932* (Typh.)
Sheppard, C. G., 220* (Tox.)
Shepperson, J. R., 643* (Hel.)
Sherman, M., (1057) (Haem.)
Shershnev, P. A., 720* (Pl.)
Sheveleva, O. N., 522* (Typh.)
Shibata, E., 467* (Hel.)
Shifrin, I. A., 819 (Typh.)
Shigeyasu, M., 182* (Hel.)
Shimansky, S. F., 765 (Tox.)
Shimizu, M., 745 (Hel.)
Shimojo, H., 602* (Typh.)
Shingai, H., 246* (Ent.)
Shipitsina, N. K., 686 (Mal.)
Shirai, M., 989* (Ent.)
Shirasaka, R., 332* (Hel.)
Shiroishi, T., 806*, 808* (Mal.)
Shishido, A., 934 (Typh.)
Shkurko, E. D., 299* (Chl.)
Shkurko, Y. D., 720* (Pl.)
Shoji, H., 466* (Hel.)
Short, G. M., 1140 (Lep.)
Shortt, H. E., 930 (Leish.)
Shrivastav, J. B., 447* (Lep.)
Shrivastava, J. B., 1275* (Der.)
Shubin, B. F., 541 (Lep.)
Shulov, A., 654, (1058), 1058* (Vms.)
Shustrov, A. K., 836 (R.F.)
Shute, G. T., 272* (Mal.)
Siboulet, A., 1063 (Misc. Dis.)
Sibthorpe, E. M., 1180* (Haem.)
Sicart, M., 270* (Mal.)
Sidorova, G. A., 287* (Leish.)
Siedlecki, E., 1047 (Hel.)
Sigalas, R., (954) (Hel.)
Sigall Montenegro, O., 623 (Lep.)
Silberstein, W., 1024 (Rab.)
Sileanu, L., 754* (Hel.)
Silich, V. A., 147, 711* (Typh.)

* *joint author*

Silva, C., 1141 (Lep.)
Silva Júnior, M., 995 (B.R.)
Silverie, R., 280* (Tryp.)
Silverman, P. H., (550) (Hel.)
Silvestroni, E., (970) (Haem.)
Simic, M., 707* (Typh.)
Simionescu, O., (885)* (Parasit.)
Simitch, T., 767, 768 (Tox.)
Simmons, S. W., 903 (B.R.)
Simpson, D. A., 1283 (Misc. Dis.)
Simpson, H. R., 811* (Tryp.)
Simson, I., 884* (Misc. Dis.)
Singer, I., 136* (Tryp.)
Singh, A., 662 (Misc. Dis.)
Singh, H., 596* (Mal.)
Singh, K. R. P., 244, 370* (Ent.)
Singh, M. V., 594* (Mal.)
Singh, N., 1106* (Mal.)
Singh, N. N., 778* (Ent.), 805* (Mal.)
Singha, H. S. K., 1260 (Hel.)
Sinnecker, H., 103 (Parasit.)
Sipahioglu, H., 860 (Hel.)
Sippola, V., 71* (Hel.)
Siqueira, A. F., 22* (Tryp.)
Siran, M., 7* (Mal.)
Sirett, N. E., 181* (Hel.)
Sirsi, M., 10, 17* (Mal.)
Siverly, R. E., 892 (Ent.)
Sívori, A., (353)* (Haem.)
Skieller, L., 563 (Tox.)
Sleisenger, M. H., (82)* (Sp.)
Slesarenko, V. V., 947 (R.F.)
Ślezak, S., 736* (Hel.)
Smadel, J. E., 814* (Typh.)
Smaikina, M. G., 764* (Tox.)
Smetana, H. F., 563* (Tox.)
Smit, F. G. A. M., 438 (Pl.)
Smit, Z. M., 474* (Def. Dis.)
Smith, A., 410, 515, 917, 1105 (Mal.), 990 (Ent.)
Smith, C. C., 691 (Mal.)
Smith, C. E., 1209 (B.R.)
Smith, C. E. G., 252 (Reports, etc.)
Smith, C. H., 353*, 652*, (763)* (Haem.)
Smith C. N., 893*, 987*, 1286* (Ent.)
Smith, D. J. W., 1019* (Typh.)
Smith, E. W., 651 (Haem.)
Smith, F. M., 61 (Hel.)
Smith, I. M., 430* (Typh.), (520), (927) (Tryp.)
Smith, J. L., 825*, 1027 (Pl.)
Smith, J. T., 734* (Lep.)
Smith, K. V., (476) (Sp.)
Smith, P. N., 721, 826 *bis* (Pl.)
Smith, R. S., 626 (Lep.)
Smith, S. A., 469* (Hel.)
Smith, W. W., 886* (Ent.)
Smithers, S. R., 319 (Hel.)
Smorodintsev, A., 252 (Reports, etc.)
Smythe, P. M., 346 (Def. Dis.)
Snyder, J. C., 253 (Reports, etc.)
Soap, 992 *quat.* (Ent.)
Soares, R., 412 (Mal.)
Soares, R. M., 412*, 413* (Mal.)
Soave, O. A., 655 (Tox.)
Soberón y Parra, G., 689 *bis* (Mal.)
Sobral, F. da C., 1246* (Lep.)
Sobrinho, J. M. A., (1206)* (Ent.)
Soc. Belge de Méd. Trop.: Nederl. Vereniging v. Trop. Geneesk, 556 (Def. Dis.)
Söderhjelm, L., 82* (Sp.)
Soeiro, A., 256 (Reports, etc.), 1110 (Mal.)
Soeroto, R. M., 7* (Mal.)
Sogani, R. K., 834* (Am.)
Sokhey, S. S., 1209 (B.R.)
Sokolinsky, I. R., 1268 (Hel.)
Sokolova, N. F., 430, 711, 711* *bis* (Typh.)
Solente, G., 167* (Ys.)
Solomon, H., (1227)* (Typh.)
Solov'ev, V. D., 531 (Rab.)
Sölvell, L., 872 (Sp.)
Soman, D. W., 530* (Rab.)
Somers, G. F., 850 (Hel.)
Somerset, E. J., 305 (Lep.)
Somov, G. P., 523 (Typh.)
Somova, A. G., 1119 (Typh.)
Soper, F. L., 257 (Reports, etc.), 294 (Y.F.)
Soresco, A., 335, 962* (Hel.)
Sotelo, C., 841*, 1144* (Lep.)
Soto, R., 551* (Hel.)
Sotolongo Guerra, F., (170) (Hel.)
South Pacific Commission, 75, 1166 (Hel.), 200, 472, (Def. Dis.) 1035 (Lep.)
Sowa, J., 95* (Oph.)
Spalatin, J., 145* (Typh.)
Sparrow, H., 302 (R.F.)
Spencer, I. W. F., 765 (Tox.)
Spits-Eshuis, W. C., 1188* (Tox.)
Spooner, D. F., 1113* (Tryp.)
Sprague, C. C., 1056* (Haem.)
Squibb, R. L., (758)* (Def. Dis.)
Squire, F. A., 925 (Tryp.)
Squires, H. C., 380 (B.R.)
Sretenovic, B., 754* (Hel.)
Srichaikul, T., 920 (Mal.)
Srikantia, S. G., 1178 (Def. Dis.)
Srinivasan, M., 994* (B.R.)
Stam, A. B., 182* (Hel.)
Stamm, W. P., 253 (Reports, etc.)
Stam-Mielziner, S. J., 182* (Hel.)
Standard, K. L., (867), 1050 (Def. Dis.)
Standen, H., 1203* (Ent.)
Standen, O. D., 639* (Hel.)
Standfast, H. A., 1110* (Mal.)
Staninec, M., 617 (Am.)
Stark, H. E., 533* (Pl.)
Starke, G., 433 (Rab.)
Starkoff, O., 672 (Ent.)
Starzyk, J., 1186 (Tox.)
Stauber, L. A., 239 (Parasit.), 427 (Leish.)
Stein, H., 1178* (Def. Dis.)
Steinbach, M., (885)* (Parasit.)
Steiniger, F., 823 (Rab.)
Stephen, L. E., (520) *bis* (Tryp.)
Sterman, M. M., 748 (Hel.)
Stern, G., 652* (Haem.)
Steyn, J. J., 1219 (Mal.)
Stirewalt, M. A., 848*, (1041) (Hel.)
Stoenner, H. G., 1121 (Typh.)
Stoker, M. G. P., 144* (Typh.)
Stoll, N. A., 240 (Parasit.)
Stoll, W. G., (566)* (Misc. Dis.)
Stolze, E., (228) (Misc. Dis.)
Stone, M. M., 729*, 1037* (Lep.)
Strauss, A., 771* (Vms.)
Strejan, G., (630) (Hel.)
Stroczyńska, M., 300 (Am.)
Strukov, A. I., 764 (Tox.)
Stubber, L. A., 363* (Misc. Dis.)
Suarez, R. M., 1055 (Haem.)
Subbotin, A. A., 711 (Typh.)
Subrahmanyan, V., 344*, 345, 966* (Def. Dis.), (774)* (Misc. Dis.), 994* (B.R.)
Subramaniam, H., 190, 468 (Hel.)
Suessenguth, H., 1269 (Hel.)
Sugai, K., (949) (Lep.)
Sugawara, K., 466*, 859 (Hel.)
Sugiyama, H., 815* (Typh.)
Suidan, F. G., 856* (Hel.)
Suitor, E. C. Jr., 707* (Typh.)
Sukumaran, P. K., 89* (Haem.)
Sumanam, S. D., 468* (Hel.)
Sun, C. C., 326* (Hel.)
Sun, Q. X., (1159) (Hel.)
Sundararaman, S., 7, 275 (Mal.)
Sur, A. M., 202* (Def. Dis.)
Sureau, P., (1121)* (Typh.) (1125) (Rab.), (1202)* (Ent.)
Surgalla, M. J., 40*, 154*, 437*, 824* (Pl.)
Surtees, G., 107, 668, 887 (Ent.)
Susanna, L., 749 (Hel.)
Sushida, K., 56*, 845, 950, 1149* *bis* (Lep.)
Susman, I. A., 452 (Lep.)
Suzuki, A., 659* (Oph.)
Suzuki, T., 989 (Ent.)
Svet-Moldavskaya, I. A., 295, 607 (Rab.)
Svet-Moldavskii, G. R., (296) (Rab.)
Swaminathan, M., 344*, 345*, 966* (Def. Dis.), (774)* (Misc. Dis.)
Swaminathan, M. C., 1177* (Def. Dis.)
Swarup, S., 1177* (Def. Dis.)
Swellengrebel, N. H., 439 (Am.), 1107 (Mal.)
Symonds, B. E. R., 202 (Def. Dis.)
Szégli, L., (935)*, 1227* (Typh.)
Szumlewicz, A. P., 250 (Reports, etc.)

T

Tabolin, V. A., 764 (Tox.)
Tagle V., I., (1069) (Ent.)
Tahori, A. S., 881* (Der.)
Tai, S. Y., 799* (Mal.)
Tailyour, J. M., 1019* (Typh.)

* *joint author*

Taiwan Provincial Malaria Res. Inst., 415 (Mal.)
Takahashi, S., (890) (Ent.)
Takaishi, K., 1131 (Chl.)
Takano, S., 1199* (Parasit.)
Takei, H., 775* (Parasit.)
Takemura, S., (218)* (Vms.)
Takeshita, Y., (853)* (Hel.)
Takeuchi, M., (853)* (Hel.)
Taliaferro, W. H., 237 (Parasit.)
Tálice, R. V., 356 (Tox.)
Tampi, M. R. V., 190* (Hel.)
Tanaka, H., 332*, 333, 1253* (Hel.), 943 (Pl.)
Tanaka, R., 815 (Typh.)
Tanaka, S., 628* (Lep.)
Tanamal, S. T. W., 722 (Chl.)
Tanase, Y., (272)* (Mal.)
Tang, F. F., 565 (Oph.)
T'Ang, H. C., 484* (Heat Str.)
Tang, J. Y., 807* (Mal.)
Tani, T., 628* (Lep.), 1199 (Parasit.)
Tanner, R. E. S., 900 (Misc. Pap.)
Tan Phui Yong, 1048* (Def. Dis.)
Tarabini C., G., (304), 446, 446*, 452, 842 *bis*, 844 (Lep.)
Tarabini, G., 1144 (Lep.)
Tarabini, J., (843)* (Lep.)
Tarasevich, I. V., 28 (Typh.)
Tarlov, A., (10)* (Mal.)
Tartari, G., (962) (Hel.)
Tasker, P. W. G., 478, 873, 1054 (Haem.), 638 (Hel.)
Tat, L., 620* (Lep.)
Tatarinov, A. L., 959 (Hel.)
Tate, J., 731* (Lep.)
Tateno, S., 934* (Typh.)
Taufflieb, R., (1205) (Ent.)
Tăutu, L., 410* (Mal.)
Tavares, B. M., 1015* (Tryp.)
Tav'ev, B. M., 819* (Typh.)
Tay, J., 548* (Hel.)
Taylor, E. P., 553* (Hel.)
Taylor, R. M., 252 (Reports, etc.)
Tedeschi, G., 615* (Am.)
Teesdale, C., 986 (Ent.)
Tejada, A., 1013 (Tryp.)
Teng, Y. T., (463)* (Hel.)
Teramura, S., 815* (Typh.)
Teravskii, I. K., 836 (R.F.)
Terencio, J., 446* *bis*, 452* (Lep.)
Terencio de las Aguas, J., (305), 842 (Lep.)
Teriteanu, E., 410* (Mal.)
Terry, R. J., 553* (Hel.)
Terzian, L. A., (987)* (Ent.)
Thangaraj, R. H., 305, 730 (Lep.)
Thannisch, G., 186 (Hel.)
Theiler, G., 1289* (Ent.)
Theiler, M., 606 (Y.F.)
Theron, J. J., 348* (Def. Dis.), 649 (Haem.)
Thiele, H. G., (746) (Hel.)
Thiermann, E., 357, 881 (Tox.)
Thiodet, J., 37 (Rab.), 1063 (Misc. Dis.)
Thompson, M. J., 927* (Tryp.)
Thompson, P. E., 130 (Mal.)
Thomson, W. E. F., 423* (Tryp.)
Thonard, J. C., 953* (Hel.)
Thonnard-Neumann, E., 559* (Def. Dis.)
Thorsteinson, A. J., (110) (Ent.)
Thumala, A., 1136* (Am.)
Thurston, J. P., 281 (Tryp.)
Tigano, F., 835* (Am.)
Tiglao, T. V., 254 (Reports, etc.)
Tilley, J. B., (304)* (R.F.)
Timms, A. R., 848 (Hel.)
Timms, G. L., 816 (Typh.)
Timofeeva, M. M., (709)* (Typh.)
Ting, K. S., 636*, (1159)* (Hel.)
Tinker, M. E., 431* (Y.F.)
Tin Tin Oo, (559)* (Def. Dis.)
Tjalma, R. A., 430 (Typh.)
Tobie, E. J., 136, 926*, 927* (Tryp.)
Toda, N., 943* (Pl.)
Toda, T., 951 (Lep.)
Todd, K. W., 1280 (Misc. Dis.)
Tokareva, A. A., 720* (Pl.)
Tokunaga, T., 951* (Lep.)
Tolentino, J. G., 844* (Lep.)
Tomat, E., (1031)* (Am.)
Tong, J. L., 482 (Der.)
Tonge, J. I., 936 (Typh.)
Törnblom, N., 82* (Sp.)
Torrealba, J. F., 462 (Hel.)
Torres, C. M., 1015 (Typh.)
Torres, E., 48*, 49* (Am.)
Torroella, J., 78 (Hel.)
Toschi, G., (487) (Ent.)
Toshioka, S., 779* (Ent.)
Tos-Luty, S., 1064* (Parasit.)
Toumanoff, C., 509, 1104 (Mal.), 1263 (Hel.)
Tousimis, A. J., (101)* (Misc. Dis.)
Trager, W., 278, 1111 (Mal.)
Tram, L., 215* (Haem.)
Tran Van Bang, 1038* (Lep.)
Trapet, P., 1030* (Am.)
Trappmann, R., 622 (Lep.)
Tréguier, J., (953)* (Hel.)
Tresca, G., 1239* (R.F.)
Trespalacios, F., (1191)* (Der.)
Trevisani, A., (962)* (Hel.)
Trincão, C., 254 (Reports, etc.), 516 (Haem.), 1116 (Tryp.)
Trinidad Govt., 417 (Mal.)
Triozon, F., 854*, 1256 (Hel.)
Tripod, J., 732* (Lep.)
Tripodi, P., (1193) (Ulc.)
Trobaugh, F. E., Jr., 1183* (Haem.)
Tromba, F. G., (962) (Hel.)
Trop. & Geograph. Med., (844) (Lep.)
Trowell, H. C., 901 (B.R.)
Truba, I. V., 819* (Typh.)
Tsang, K. C., 99* (Misc. Dis.)
Tseng, H. L., 762 (Haem.)
Tsou, H. W., 324 (Hel.)
Tsuge, K., 628 (Lep.)
Tsutsui, J., 1276 (Oph.)
Tsutsumi, C., (991) (Ent.)
Tu, C. K., 1158* (Hel.)
Tublin, I., 973* (Haem.)
Tucker, B., 111* (Misc. Pap.)
T'ung, S. T., 1165 (Hel.)
Tung, T. C., 1054* (Haem.)
Turner, L. H., 551*, 862 (Hel.)
Turner, P. P., (1196) (Misc. Dis.)
Tverdy, G., 198* (Hel.)
Twohy, D. W., 106*((Ent.)

U

Uilenberg, G., (926) (Tryp.)
Ulrich, A. M., 516* (Mal.)
Uminski, J., 1064* (Parasit.)
Undavia, J. V., 874 (Haem.)
Ungureanu, E., 1291 (Ent.)
Ungureanu, L., 1291* (Ent.)
U.S. Dept. of Health, Education, and Welfare, 45 (Am.)
U.S. Pub. Health Service, 991 (Ent.)
Ureles, A. L., 187 (Hel.)

V

Vaandrager, K., 610 (Am.)
Vachhrajani, R. B., 873 (Haem.)
Vail, J. W., 1105* (Mal.)
Vaillant, 146* (Typh.)
Vainshtein, M. A., 817 (Typh.)
Vaishwanar, P. S., 872 (Haem.)
Vake, A., 609 (Pl.)
Valade, M., (989)* (Ent.)
Valdez de Macías, A. M., 948* (Ys.)
Valensi, A. L., 252 (Reports, etc.)
Valentine, E. H., 482* (Der.)
Van Asperen, K., 778 (Ent.)
Van Bogaert, L., 198 (Hel.)
Vanbreuseghem, R., 252 (Reports, etc.), 979, 1275 (Der.)
Van Dam-Bakker, A. W. I., (579)* (Misc. Pap.), 1048* (Def. Dis.)
Van den Akker, S., 1188 (Tox.)
Van den Assem, J., 411, 1221 (Mal.), 489 (Ent.)
Van den Berghe, L., 253 (Reports, etc.), 560 (Def. Dis.), 696, 697 (Mal.), 700 (Tryp.)
Van den Dorpe, A., 643 (Hel.)
Vandeputte, M., 100 (Misc. Dis.), 1275* (Der.)
Vanderborght, H., 347*, 349* (Def. Dis.)
Van der Hoeven, J. A., 81 (Def. Dis.)
Vanderick, F., 412* (Mal.)
Van der Kuyp, E., 147 (Y.F.)
Van der Sar, A., 1151* (Hel.)
Van der Schalie, H., 319 (Hel.)
Van Dijk, M. C., (876) (Haem.)
Van Dijk, W. J. O. M., 411*, 1009 (Mal.) 1167* (Hel.)
Van Gorkum, A. E. H., (1138) (Lep.)
Vanier, G., 833* (Am.)

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Van Keulen, A., 955 (Hel.)
Van Rensburg, L. C. J., 187 (Hel.)
Van Riel, J., 379 (B.R.)
Van Sande, M., 19*, 1225* (Tryp.)
Van Someren, E. C. C., 487 (Ent.)
Van Thiel, P. H., 93 (Tox.), 182* (Hel.), 829* (Am.)
Van Zanen, G. E., 1031 (Ys.)
Varela, G., 251 (Reports, etc.), 480, 1060 (Tox.)
Vargas, L., (31) (Y.F.), 109, 669, 672 (Ent.), 416, 689, 802 (Mal.)
Vasconcelos, M. E. B., 255 (Reports, etc.)
Vasil'eva, L. D., 817* (Typh.)
Vasina, S. G., 764, 769 (Tox.)
Vaz, J. M., 1194 (Misc. Dis.)
Vázquez, J., 551* (Hel.)
Veaux, R., (954)* (Hel.)
Veeger, L. M., 1297 (Reports, etc.)
Veeraraghavan, N., 716 (Rab.)
Vegas, M., 52 (Lep.)
Veiga, A. A., 138 (Tryp.)
Veitieka, J., 1036* (Lep.)
Velame, V. P., 1151* (Hel.)
Velasco C., F. J., 1179 (Def. Dis.)
Velasco F., O., 939 (Rab.)
Velazquez Gaztelu, F., 689* (Mal.)
Velickovic, C., 754* (Hel.)
Vella, E. E., 684 (Mal.)
Vella, F., 88, 561, 650 (Haem.)
Vellieux, M., 658, 772 (Oph.)
Veloso, H. P., 802, 1106 (Mal.)
Venkat Rao, V., (16) (Mal.)
Venter, E. E., 966* (Def. Dis.)
Ventura, S., 761* (Haem.)
Verano, O. T., 21*, 139* (Tryp.), (118)* (Mal.), (1205) (Ent.)
Vereninova, N. K., 1134* (Chl.)
Vervoorn, J. D., (249) (Misc. Pap.), 630 (Hel.), 1194 (Misc. Dis.)
Vesenjak, J., 145 (Typh.)
Viazhevich, V. K., (153) (Rab.)
Vidales, H., 1020* (Y.F.)
Vidrine, A., Jr., (186)*, (1259)* (Hel.)
Viel, B., 1243 (Lep.)
Vieto, P. L., 170* (Hel.), 424* (Tryp.)
Villari, V., 824 (Rab.)
Villemot, J. M., 1023 (Rab.)
Vincent, M., 557, 871* (Def. Dis.)
Vinke, B., 1151 (Hel.)
Vis, H., 871 (Def. Dis.)
Viteri, F., 205*, (758)*, 966*, 967* *bis* (Def. Dis.)
Vodička, L., 145* (Typh.)
Voelckel, J., 43 (Pl.), 1108 (Mal.)
Voiculescu, R., (935)*, 1227* (Typh.)
Voisin, H., 76* (Hel.)
Volovich, N. I., 434 (Rab.)
Von Brand, T., 926, 927 (Tryp.)
Vondráčková, A., 1126 (Rab.)
Von Fintel, H., 42* (Pl.)
Von Harnack, G. A., 1042 (Hel.)
Von Hofsten, C. G., 670* (Ent.)
Von Holt, C., 484, 1194, 1194* (Misc. Dis.)
Von Holt, L., 1194 (Misc. Dis.)
Vora, D. D., 1151 (Hel.)
Vortel, V., 1126* (Rab.)
Vosloo, A., 1219* (Mal.)
Vošta, J., 736 (Hel.)
Vostál, Z., 893 (Ent.)
Vouilloux, P., 1030 (Am.)
Voyt, E. B., 764, 769* (Tox.)
Vuillet, F., (1202)* (Ent.)
Vukasović, P., 1008 (Mal.)
Vukobratovic, S., 1184* (Vms.)
Vukotic, M., 1008* (Mal.)
Vulcan, P., (838) (Lep.)
Vyas, K. J., 761 (Haem.), 1293 (Misc. Pap.)
Výmola, F., 1126* (Rab.)

W

Wable, S. M., 448* (Lep.)
Waddy, B. B., 255 (Reports, etc.)
Wade, H. W., 1241* (Lep.)
Wadham, B. McM., 679 (Reports, etc.)
Wadsworth, G. R., 867, 1048* (Def. Dis.)
Wagenvoort, C. A., 829 (Am.)
Wagner, E. D., 544, 851*, 1041 (Hel.)
Walden, B., 353* (Haem.)
Wallace, G. A., 1232* (Rab.)
Wallace, H. J., 619* (Ys.)
Wallace, J. H., 306, 734* (Lep.)
Walls, K., 1192* (Der.)
Walls, L. P., 25* (Leish.)
Walsh, R. J., 476*, 1181 (Haem.)
Walt, F., 253 (Reports, etc.), 1178 (Def. Dis.)
Walters, J., 1280 (Misc. Dis.)
Walton, A. C., 102, (885) (Parasit.)
Walton, B. C., 463*, 852, 852*, 1254* (Hel.), 702 (Tryp.)
Walton, G. A., 258 (Reports, etc.), 372, 491 (Ent.)
Wang, C. F., 339 (Hel.)
Wang, C. W., 70* (Hel.)
Wang, C. Y., 857 (Hel.)
Wang, H. C., 799* (Mal.)
Wang, S. P., (199)* (Hel.)
Wang, W. L., 327 (Hel.)
Wang, Y. L., (228) (Misc. Dis.)
Wanson, M., 198* (Hel.)
Ward, J. R., 935* (Typh.)
Warda, L., 1173 (Hel.)
Wardekar, R. V., 1246 (Lep.)
Ware, G. W., 245 (Ent.)
Warren, K. S., 635 (Hel.)
Warren, L. G., 1225 (Tryp.)
Washbourn, K., (281)* (Tryp.)
Wassermann, H. P., (212) (Haem.)
Watanabe, Y., 157*, 158 (Chl.)
Waterlow, J. C., 253 (Reports, etc.), 1050* (Def. Dis.)
Watson, H. J. C., 282 (Tryp.)
Watson, J. M., 60, 314 (Hel.)
Wattal, B. L., 117* (Mal.), 243*, (667) (Ent.)
Weaver, L. C., 49* (Am.)
Webb, J. E., Jr., 1069 (Ent.)
Webbe, G., 170, 952 (Hel.)
Webster, G. A., 854 (Hel.)
Wegmann, T., 549 (Hel.)
Wegner, Z., 6* (Mal.), 532 (Pl.)
Wei, W. P., (69) (Hel.)
Weidhaas, D. E., 666, 666* (Ent.)
Weimer, H. E., (200) (Hel.)
Weiner, D., (480) (Tox.)
Weiner, M. A., 1143 (Lep.)
Weinman, D., 563 (Tox.)
Weiser, J., 919* (Mal.), 1069 (Ent.)
Weisman, A., 654* (Vms.)
Weiss, C., (975)* (Vms.)
Weiss, E., 707 (Typh.)
Weissman, A., 1058 (Vms.)
Weitz, B., 106* (Ent.)
Welch, J. R., (112) (B.R.)
Weller, T. H., 546* (Hel.)
Wells, R., 649 (Haem.)
Welsh, J. F., 955* (Hel.)
Went, L. N., 212, 213*, 1183 (Haem.)
Werner, H., 134 (Tryp.)
Wertman, K., 146* (Typh.)
Wessman, G. E., 154 (Pl.)
West, A. S., 488* (Ent.)
West African Council for Medical Research, 579 (Reports, etc.)
West African Institute for Trypanosomiasis Research, 1011 (Tryp.)
Westall, R. G., 647 (Def. Dis.)
Weston, J. K., 130* (Mal.)
Weston, K., 130* (Mal.)
Westphal, A., (220) (Tox.)
Weyer, F., 291, 1015 (Typh.), 515*, 1107* (Mal.)
Wharton, R. H., 191, 191*, 551*, 1168 (Hel.), 251* (Reports, etc.)
White, J. C., 254* (Reports, etc.)
White, P. J., 331 (Hel.)
Whittet, T. D., 850* (Hel.)
Wibaut-Isebree Moens, N. L., (71) (Hel.)
Wiener, S., 1058, 1185 (Vms.)
Wigglesworth, V. B., (578) (Ent.)
Wijers, D. J. B., 197* (Hel.), 597, 1113, 1223* (Tryp.)
Wilcocks, C., 780 (Misc. Pap.), 1280 (Misc. Dis.)
Wild, H., 1208 (Misc. Pap)
Wilgram, G. F., 350 (Def. Dis.)
Wilkinson, G. K., 81 (Def. Dis.)
Willett, K. C., 698* (Tryp.)
Williams, C. D., 557 (Def. Dis.)
Williams, J. E., 463*, 852* (Hel.)
Williams, K., (1168)* (Hel.)
Williams, M. C., 106 (Ent.)
Williams, R. C., (82)* (Sp.)
Williams, R. R., (1176) (Def. Dis.)
Williams, R. W., 779 (Ent.)

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Williamson, J., 282*, (520)* (Tryp.)
Willis, M. J., 368* (Ent.)
Wills, V. G., 1050* (Def. Dis.)
Wilmot, A. J., 444, 832* (Am.)
Wilson, D. B., 588, 1219* (Mal.)
Wilson, G. M., 482* (Der.)
Wilson, H. G., 490*, 670*, 893 (Ent.)
Wilson, J. L., 1070 (Misc. Pap.)
Wilson, T., 121 (Mal.), 191*, 551 (Hel.), 250 (Reports, etc.)
Wilson-Jones, K., 1203 (Ent.)
Winkler, L. R., 851 (Hel.)
Winn, M. M., 852* (Hel.)
Winslow, D. J., 985 (Parasit.)
Wirth, W. W., (1202) (Ent.)
Wishahy, A. G., 645 (Def. Dis.)
Wisseman, C. L., Jr., 289* (Typh.)
Wojciechowski, E., 1018 (Typh.)
Wolfe, L. S., 574, (889), (1287)* (Ent.)
Wolfe, P. A., 723* (Am.)
Wolff, F., 1256 (Hel.)
Wolff, H. L., (496) (Reports, etc.)
Wolff, J. W., 525 (Y.F.)
Won, W. D., 39 (Pl.)
Wone, C., (954)* (Hel.)
Wong, K. C., 565* (Oph.)
Wood, J. E., 717* (Rab.)
Woodcock, R. C., 642* (Hel.)
Woodruff, A. W., 254 (Reports, etc.), 930 (Leish.), 1280 (Misc. Dis.)
Woods, R. W., 1040* (Hel.)
Woodward, S. F., 595* (Mal.)
Work, T. H., 252 (Reports, etc.)
World Health Organization, 383, (B.R.), 414, 415, 921 (Mal.), 491 (Ent.), 556 (Def. Dis.), 941 (Pl.)
WHO Chronicle, 737 (Hel.)
Worsfold, J. T., 445 (Lep.)
Worthington, E. B., 996 (B.R.)
Wragg, W. R., (281) (Tryp.)
Wright, C. A., 250 (Reports, etc.)
Wright, C. H., 1182* (Haem.)
Wright, F. J., 534 (Am.)
Wright, J., 373 (Misc. Pap.)
Wright, R. H., 887 (Ent.)
Wright, R. L., 813 (Typh.)
Wu, C. C., 426 (Leish.)
Wu, C. Y., 324* (Hel.), 800 (Mal.)
Wu, H. N., 484* (Heat Str.)
Wu, H. Y., 815* (Typh.)
Wu, L. T., 583 (B.R.)
Wu, Y. Y., 327* (Hel.)
Wunderlich, M., 200* (Hel.)
Wurzel, H., 974* (Vms.)
Wykoff, D. E., 233 (Parasit.), 328, 853 (Hel.)
Wysocka, F., 1064 (Parasit.)

X

Xavier, A. A., 255 (Reports, etc.)

Y

Yada, Y., 870 *bis* (Def. Dis.)
Yamada, N., 735, 950 (Lep.)
Yamada, S., 467* (Hel.)
Yamaguchi, T., 182, (328) (Hel.)
Yamamoto, T., 447, 630 (Lep.), 603 (Typh.)
Yamanaka, T., 79 (Hel.)
Yamanè, K., 602* (Typh.)
Yamasaki, T., (272) (Mal.)
Yamashita, M., 73, 1259 (Hel.)
Yanagihara, T., 185 (Hel.)
Yanagisawa, K., 58*, 949, 1244 (Lep.)
Yang, C. C., (218), 479 (Vms.)
Yang, J. Y., 745* (Hel.)
Yang, T. C., 324* (Hel.)
Yao, D. F., 807 (Mal.)
Yao, S. C., 326* (Hel.)
Yasukawa, T., 1149* (Lep.)
Yen, H. C., 484 (Heat Str.)
Yeo, D., 811 (Tryp.), 1219 (Mal.)
Yeoh Oon Swee, (561)* (Haem.)
Ying, Y. Y., 324* (Hel.)
Yli-Pohja, M., 101, 101* (Misc. Dis.)
Yoshino, K., 151 (Rab.), (328)* (Hel.)
Young, K., 663 (Misc. Dis.)
Young, M. D., 122*, 690, 691*, 1007* (Mal.) 750, 752 (Hel.), 833 (Am.)
Young, W. B., 884* (Misc. Dis.)
Ytrehus, K., 85 (Haem.)
Yü, C. F., 70* (Hel.), 477 (Haem.)
Yü, E. S., 815 (Typh.)
Yü, M. H., (199)* (Hel.)
Yü, P. J., (463)* (Hel.)
Yuan, H. H., (227) (Misc. Dis.)
Yuan, P. H., 484* (Heat Str.)
Yugami, S., 1260* (Hel.)
Yurkovsky, A. M., 1232 (Rab.)

Z

Zabugina, F. D., 764 (Tox.)
Zaghi, A., 696* (Mal.)
Zago, H., Jr., 73 (Hel.)
Zakaria, H., 546, 951 (Hel.)
Zambelli, P., 615* (Am.)
Zangaglia, O., 102 (Parasit.)
Zannos-Mariolea, L., 1272* (Haem.)
Zanuttini, G., 846 (Hel.)
Zarnea, G., (935), 1227 (Typh.)
Żarnowski, E., (1196) (Parasit.)
Zasukhin, D. N., 763, 764*, 765 *ter* (Tox.), 993* (Misc. Pap.)
Zdrodovski, P., 253 (Reports, etc.)
Zdrodovskii, P. F., 602, (602), 708 (Typh.)
Zeledón, R., 424 (Tryp.)
Zeller, R. W., 513 (Mal.)
Zimmermann, W. J., 81, 866, 1268 (Hel.)
Zinsou, R., (359)* (Der.)
Ziprkowski, L., 881 (Der.)
Ziti, L. M., 455* *bis*, 456*, 457* (Lep.)
Zivković, B., 707 (Typh.)
Zobel, C. R., (1258)* (Hel.)
Zonchello, S., 304* (Lep.)
Zorikhina, V. I., 956* (Hel.)
Zubkova, R. I., 292, 1121* (Typh.)
Zubok, L. P., (143)* (Typh.)
Zuckerman, A., 696 (Mal.)
Zuelzer, W. W., 650*, 971*, 1181* (Haem.)
Zufarov, T. Z., 273* (Mal.)
Zuidema, P. J., 899 (Misc. Pap.), 1050 (Def. Dis.)
Zwierz, C., 736* (Hel.)

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